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THE EARLY DIAGNOSIS OF CANCER

F. A. O'HAGAN-WARD, L.R.C.P.I., L.R.C.S.I.

South African Institute for Medical Research, Port Elizabeth

Whilst every specimen removed at operation is not submitted for pathological examination, the experience in this laboratory, being the only one of its kind in the Eastern Province, gives a fairly accurate idea of the relative incidence of cancer in this area. Table I shows the common cancers

TABLE I. CANCER, IN ORDER OF FREQUENCY, SEEN AT THE S.A.I.M.R., PORT ELIZABETH, DURING 1957

Organ	*Number of specimens seen*	*Organ*	*Number of specimens seen*
Skin	90	Stomach	24
Prostate	66	Rectum	21
Cervix	62	Urinary bladder ..	16
Breast	60	Endometrium ..	16
Oesophagus (and pharynx)	25	Ovaries	16
		Lungs	14
Colon	25		

encountered, in order of frequency, during the year 1957. As these are the common cancers in any locality, although not necessarily in this order, it seems worth while to review the features which would lead to their early diagnosis.

Cancer of the Skin

This may begin as a senile keratosis, a leukoplakia, a papule or a verrucous lesion which later ulcerates. It may arise from a lentigo or a pigmented naevus, or it may arise from apparently normal skin. Experience in this laboratory shows that the majority of cases are correctly diagnosed on clinical grounds. Because, however, there are some lesions with which it might be confused (chronic non-specific ulcer, molluscum sebaceum etc.) a specimen should be submitted for histological confirmation.

Cancer of the Prostate Gland

From a consideration of the writings of various authors, the experience of others and our own experience, surprisingly few generally accepted facts emerge. This much however seems clear:

1. It is practically impossible to diagnose macroscopically cancer of the prostate in the early stages. Though we examine more than 150 prostate glands a year we are seldom able to give an opinion as to malignancy on the basis of size, shape, colour, contour or consistency.

2. In a high percentage of cases it is exceedingly difficult to diagnose cancer of the prostate microscopically. These difficulties are not due to a malignant appearance which is in fact benign, but rather to a benign appearance which is in fact malignant.

3. Pathologists differ in what they believe are the criteria of malignancy. Hence one pathologist may report a gland benign, whilst another will report the same gland malignant.

4. It is an exceedingly common condition; some authors state that it occurs in 30% of men over 50 years of age.[1]

5. There are two distinct biological types of cancer of the prostate. One is the latent type and is often discovered as a coincident lesion in men who die from other causes. The other is active, infiltrating and forming metastases. There is no morphological means of distinguishing between the latent and the active form.

6. The most important clinical symptoms of cancer of the prostate are those grouped under the term prostatism. The most important clinical sign is hardness of the gland on rectal examination.

7. The serum-acid-phosphatase estimation is of little or no value in the early diagnosis of cancer of the prostate, but is important if there are secondary growths.

8. Cancer of the prostate may be present in a gland of normal size.

In view of all this, what should be the attitude of the general practitioner? The symptoms of prostatism and the finding of a hard prostate gland, whether that hardness be diffuse or patchy, should warrant a provisional diagnosis of prostatic cancer. Some such cases will prove to be cases of stone, fibrosis, or some other benign lesion, but such errors are of little or no importance. Cases which are provisionally diagnosed on the basis of prostatism and a hard prostate demand assessment by a skilled urologist.

Cancer of the Cervix

The first symptom is usually vaginal bleeding. This is not severe in the early stages and is often brought about by trauma, especially coitus. A watery or blood-stained vaginal discharge is another important early feature. Either of these demand a thorough gynaecological examination. Schiller's test is in no way diagnostic but it may indicate the portion of the cervix from which a biopsy should be taken.

Clinicians are so conscious of the incidence of cancer of the cervix that cervical-biopsy specimens are amongst the more common specimens received at this laboratory. The diagnosis of cervical cancer is thus on a secure basis. It has, however, been shown that a vaginal or cervical smear will show some cases which biopsy failed to show. Ideally the smear method should be used in conjunction with biopsy in those cases where cancer is suspected, and alone in cases

where it is not suspected. This, however, might be regarded as superfluous and as not justifying the trouble, time and expense involved. Obviously, if vaginal smears were sent from every woman not suspected of having cancer of the cervix, there would be little time for anything else in the laboratory.

At the present time it would seem that the best plan is to submit a biopsy specimen in suspected cases together with smears.

Cancer of the Breast

The voluminous literature on the subject of the diagnosis of cancer of the breast can be summarized as follows. Every solitary mass in the breast of a woman in the cancer age should be examined histologically. Experience in this laboratory has shown the value of this attitude. The histological examination is best performed by the frozen-section method during the operation so that, in the event of the mass proving malignant, the surgeon may carry on accordingly, and thus save the inconvenience and expense of a possible second operation.

If every breast mass in a woman of the cancer age is regarded as malignant a considerable number of errors will be made. Thus some specimens will prove to be simple hyperplasia, simple tumours, plasma-cell mastitis, and solitary cysts. Such errors however are of no consequence. The importance of discovering a cancer more than balances the inconvenience and anxiety caused by a false-positive diagnosis of malignancy.

Microscopic examination of a discharge from the nipple, when this is present, may be important in diagnosis, and it will be of the greatest importance when a mass cannot be found on palpation. In practice, however, it is found that only a small proportion of breast cancers present this feature; hence it is not of great diagnostic importance.

The surgeon who would treat cancer of the breast must be confident of the diagnosis. This confidence can only be obtained in many cases if a histological examination is made.

Cancer of the Oesophagus

The outstanding symptom of cancer of the oesophagus is dysphagia. This is slowly progressive and occurs first for solids and later for liquids. The other features of the disease, such as retrosternal pain, cough, recurrent-laryngeal-nerve paralysis etc., may occur quite early, but usually dysphagia dominates the clinical picture. The early diagnosis, therefore, will depend upon the thorough investigation of this symptom. This calls for the exclusion of the other well-known causes of dysphagia (Plummer-Vinson syndrome, scleroderma, dermatomyositis, aortic aneurysm etc.) and will involve a radiological examination, oesophagoscopy and possibly a cytological examination of the gastric contents.

Cancer of the Stomach

The early symptoms of cancer of the stomach are vague and no definite conclusion can be drawn from them. All that can be expected from a clinical examination is to arrive at a suspicion of its existence. Persistent dyspepsia, whether this takes the form of epigastric fullness, belching, vague abdominal discomfort, loss of appetite or vomiting—any of these in a person over the age of 40 years calls for investigation and the exclusion of stomach cancer. Several aids to diagnosis are available:

1. *The fractional test meal.* This is a time-honoured investigation but it is difficult to see what help it gives in solving the problem. It is true that some 50-60% of stomach cancers are associated with achlorhydria. But some 40-50% therefore, show no such association. Furthermore, achlorhydria is found in a number of other diseases and even in some 'normal' people. This procedure, therefore, is mentioned to be dismissed as thoroughly unreliable and possibly even misleading.

2. *Radiological examination.* This is generally regarded as the most useful procedure and should certainly be done in all suspected cases. A filling defect is the classical radiological sign, but the lesion must have progressed considerably before it will give this sign. Bamforth has reported 5 cases of cancer of the stomach all of which were unrecognizable on gross examination.[2]

3. *Gastroscopy.* This method has its advocates and in skilled hands, when combined with biopsy, it should prove the most useful method of all. We in this laboratory, however, have had no experience of it.

4. *Exfoliative cytology.* Writing in a recent number of the *British Medical Journal*, Schade[3] states his experiences in these words: 'So far about 2,500 gastric cytological examinations have been made. Twenty-five cases of carcinoma have been discovered by cytology where other investigations (radiology and gastroscopy) have been negative.' Further on he says: 'One important conclusion to be drawn from our experience is that the best material for cytology was obtained from those cases in which the malignant lesion was subsequently found to be small, often confined to the mucosa.' It seems therefore that in exfoliative cytology we have not only a great aid to diagnosis but a means of diagnosing stomach cancer at a very early stage.

Cancer of the Rectum

The majority of cases of cancer of the rectum occur in the upper third of this organ. They begin as an indurated lesion, a nodule or a plaque. Later, a deep ulcer with characteristic margins forms or the lesion develops into a cauliflower growth. The early symptoms which demand investigation are (1) the passage of blood and mucus at stool, (2) a feeling of incomplete emptying of the bowel and (3) increasing constipation, often manifested by an increasing need for aperients.

On the subject of the diagnosis of cancer of the rectum no one speaks with greater authority than Gabriel,[4] who sums the matter up in the following words: 'It may be safely stated that about 90% of rectal carcinomas can be felt digitally and, if the sigmoidoscope is used in addition, a correct diagnosis should be made in every case'.

The special investigation, therefore, in cases where cancer of the rectum is suspected, is sigmoidoscopy, with which may be included a biopsy.

Cancer of the Colon

Because of its inaccessability, cancer of the colon is exceedingly difficult and usually impossible to diagnose in the early stages. The outstanding symptom is a change in bowel habit. This may manifest itself by increasing constipation, resulting in an increasing need for aperients. Constipation which alternates with diarrhoea is another suggestive symptom. The outstanding sign is the passage of blood and mucus *per rectum*. Three things, however, must be remembered about this sign: Firstly, it is by no means pathognomonic;

secondly, it indicates that the lesion is fairly advanced; and, thirdly, it is obscure if the lesion is situated in the proximal colon. If, for example, the cancer is situated in the caecum, there will be nothing characteristic about the stool on gross examination. In this event, however, the benzidene test for occult blood will be useful. The difficulties therefore, in the early diagnosis of cancer of the colon are very great, so much so that chronic hypochromic anaemia may be, and often is, the presenting feature.

The most important aids to diagnosis at the present time are radiological examination after an opaque meal; more important, an opaque enema and sigmoidoscopic examination with which may be included a biopsy. Cases have been diagnosed by a cytological examination of colonic washings, and this method appears to have distinct value. It is, however, too early yet to state its usefulness.

Cancer of the Bladder

The outstanding clinical feature of cancer of the bladder is a painless intermittent haematuria. Any patient, therefore, in the cancer age who presents this feature must be regarded as having cancer of the bladder until a complete investigation, especially a cystoscopy, is performed. Because the haematuria is often intermittent and painless, a patient during an intermission may be reluctant to submit to a cystoscopic examination. This reluctance is understandable, but he should nevertheless be prevailed upon to submit to examination. If he still refuses recourse may be had to a cytological examination of the urine for malignant cells. This latter, however, is no substitute for a cystoscopic examination, but if positive or doubtful, it may be a strong factor in persuading the patient to have the complete examination. If followed, the procedure outlined above will bring many early cases to light.

Cancer of the Endometrium

Post-menopausal bleeding is probably the most common presenting symptom. It must be remembered, however, that cancer of the endometrium is by no means confined to post-menopausal women. Before the menopause a blood-stained vaginal discharge is an important early feature.

As with cancer of the cervix, clinicians are very conscious of the incidence of endometrial cancer. Well over 1,000 endometrial specimens are received in this laboratory every year. The vast majority of these prove to be benign; nevertheless this figure shows a high degree of suspicion on the part of the clinicians and it is thus felt that few cases are missed. Exfoliative cytology may be used as an adjunct to currettage in suspicious cases but not as a substitute.

Cancer of the Ovaries

It is not possible to diagnose early cancer of the ovaries by purely clinical means. All that can be achieved by a gynaecological examination is to arrive at a suspicion by finding an ovarian mass. Laparotomy will usually disclose the nature of the mass, but sometimes it will not; hence all specimens should be sent to the laboratory for histological examination.

Cancer of the Lung

This was the subject of an earlier paper,[5] and so nothing will be said here except that a suspicion of cancer of the lung should be investigated by radiology, bronchoscopy and exfoliative cytology.

What is badly needed is a reliable serological test for cancer. Many such tests have been proposed in the past but so far none has proved satisfactory. With the exception of cancer of the skin and rectum, we see that clinical assessment of cases leads to little more than a suspicion of the existence of cancer. For accurate diagnosis recourse must be had to special investigations which are: (1) radiological examination, (2) endoscopy (with biopsy) and (3) exfoliative cytology.

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A BANTU CASE OF HAEMOLYTIC ANAEMIA RESEMBLING ACHOLURIC JAUNDICE

F. GON, M.B., B.CH., D.T.M. & H.,

Blood Group Research and Transfusion Laboratories, South African Institute for Medical Research, Johannesburg

Acholuric jaundice is considered to be a rare condition in American Negroes. Irby[1] reported a total of 20 cases occurring in 10 families. Later Kline and Holman,[2] in a comprehensive search, collected a total of 42 *bona fide* cases with a familial history, representing 13 different family trees.

In the African this condition is considered an even rarer entity. Gelfand[3] has never seen it in an African, although Foy and Kondi[4] in an article on 'Anaemia in the African' quote one typical case with a family history. Merskey and Baskind[5] published a case of 'Chronic Haemolytic Icterus resembling Acholuric Jaundice occurring in an African male'. Their patient presented with a complaint of pain in both legs and on investigation was found to have an anaemia of which the features were identical with those of acholuric jaundice, but a familial study could not be carried out, and comprehensive techniques for the exclusion of antibodies had not been evolved at that time. The case described here also falls short of a proved example of congenital haemolytic anaemia in that a family study could not be made—a common and disappointing problem in dealing with Africans.

The patient, a young Bapedi male of about 20 years, normally resident in the Northern Transvaal, and at present employed on one of the Reef gold mines, presented himself as a donor at one of the weekly bleeding sessions carried out by the South African Institute for Medical Research. He passed the copper-sulphate specific-gravity (1053) test for haemoglobin and 500 c.c. of blood were collected without any obvious ill effects. The following day, during the course of grouping tests, it was noticed that the serum in the 'pilot-tube' appeared to be icteric, and on examination of the bottle of blood it was seen that about a quarter of the volume of settled cells had haemolysed, and after 5 days' storage at 4°C almost the total volume of cells had undergone haemolysis.

The mine medical officer was immediately notified and the patient hospitalized. Further specimens were obtained and the following investigations were carried out 6 days after the donation of blood, with the results shown:

1. Haemoglobin 11·5 g.%. Leucocyte count 5·2 thousand

per c.mm. (neutrophils 61·0%, monocytes 1·5%, eosinophils 2·0%, basophils 1·0%, late normoblasts 1·0%). Normochromia +, anisocytosis ++, poikilocytosis +, diffuse basophilia ++, spherocytosis ++, reticulocytes 23%.

2. *Serum Bilirubin.* Direct 0·6 mg. per 100 ml., total 3·1 mg. per 100 ml.

3. *Antibody Investigations.* Direct Coombs test negative, indirect Coombs test negative, trypsinized cells negative, ficinized cells negative. Cold agglutinins absent. Indirect Donath-Landsteiner reactions negative.

4. *Haemoglobins.* Alkali denaturation, solubility, and paper electrophoresis, all conformed to normal adult haemoglobin.

5. Schumm's test positive.

6. V.D.R.L. negative

7. *Urine, microscopical:* Ova of *S. haematobium* present. Red blood corpuscles. *Chemical:* Bilirubin absent, urobilin +++.

8. Malaria negative.

A fragility test could not be carried out at that time because the blood had commenced to haemolyse in the short time taken to convey the specimens from the mine to the laboratory (a distance of some 30 miles). For this investigation the patient was therefore brought to the laboratory.

He was a well-nourished young adult with no history of ill-health; he had carried out a normal day's work as an underground worker without any complaints and in fact, as he felt quite well, could not understand why he was being hospitalized. Close questioning regarding any family history of illness or jaundice yielded negative results, but as the patient was a poor witness, little importance could be attached to this. He was quite unaware that his conjunctivae were obviously jaundiced.

His liver was found to be about one finger enlarged and the edge of his spleen was just palpable.

He had a large ulcer on the anterior aspect of the lower third of his right leg, which he said had been present for some time, although he could not remember traumatizing the area. Healed scars were present on both legs and he gave a history of previous ulcers which had, apparently, appeared and healed spontaneously.

TABLE I. FRAGILITY TEST

% Saline	Control		Specimen	
	Before incubation	*After 18 hrs. at 37°C*	*Before incubation*	*After 18 hrs. at 37°C*
	%	%	%	%
0·00	100	100	100	100
0·05	100	100	100	100
0·10	100	100	100	100
0·15	100	100	100	100
0·20	99	100	100	99
0·25	98	100	100	96
0·30	95	100	96	91
0·35	91	100	92	84
0·40	85	98	86	77
0·45	71	95	77	67
0·50	35	87	55	56
0·55	10	38	16	40
0·60	4	8	6	25
0·65	2	5	2	17
0·70	0	2	0	9
0·75	0	0	0	4
0·85	0	0	0	0

A screen fragility test was then carried out, which showed haemolysis commencing in 0·72% saline and complete in 0·32%. The result of an incubated fragility test revealed an increase in haemolysis in the higher concentrations of saline as compared to a normal control (Table I). These findings are compatible with congenital spherocytosis.

COMMENT

The patient was undergoing a haemolytic process at the time of his accidental discovery. The anaemia could not be considered symptomatic in nature since no predisposing factors could be found, and treatment for bilharziasis had not been commenced. The anaemia associated with bilharzia is usually mild and of the hypochromic type. In severe cases with marked liver involvement the anaemia may be macrocytic.[3]

An extensive search for auto- and other antibodies yielded negative results; this was in favour of a congenital rather than an acquired condition.

The anaemia closely conformed to that of acholuric jaundice, but owing to the difficulties encountered with migrant African labour, a familial study for the essential confirmatory evidence could not be undertaken.

Up to 1953, 20 cases had been reported in American Negroes, and only 4 years later the total had increased to 42. In South Africa no proven case has been published to date; it is suggested, however, that a number of carriers may go undetected, since the symptoms are usually mild, so that the patient may quite often lead an active life—the only obvious sign being a slightly sallow tinge of the skin (not applicable here) and sometimes an intractable ulceration of the legs or feet the presenting and only symptom.[6]

In this case, as an example, we have a patient with obviously jaundiced conjunctivae and a chronic ulcer of the right leg, who did not consider himself in any way ill and was only discovered because he presented himself as a blood donor.

SUMMARY

The chance discovery of a case of haemolytic anaemia resembling acholuric jaundice in a young adult African is described. This patient shows that cases may go undetected because of the mildness of the symptoms and emphasizes the difficulty of obtaining corroboratory familial evidence in our South African Bantu.

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EDITORIAL : VAN DIE REDAKSIE

IRON-DEFICIENCY ANAEMIA IN CHILDHOOD

The application of the knowledge gained about vitamin-deficiency diseases and the widespread use of vitamins by child welfare clinics, hospitals, and physicians have almost eradicated these diseases. Long before the discovery of vitamins, however, iron was known to be necessary for the haemoglobinization of red blood corpuscles and known to be effective in the prevention and treatment of hypochromic anaemia. For various reasons, however, the value and efficacy of iron have on occasion been discredited.

It is surprising that, in a country such as America, where all aspects of nutrition are paramount in the minds of practising physicians, the incidence of iron-deficiency anaemia, in one centre at least, is as great today as it was two decades ago.[1] In Cape Town, Lanzkowsky and McKenzie,[2,3] in a recent article in this *Journal*, have drawn attention to the high incidence of iron-deficiency anaemia in pre-school-going children attending various crèches in the Cape Town area. They also stressed the importance of maintaining an optimal haemoglobin level especially in the under-privileged sections of the community.

The association of an iron-deficiency anaemia with certain conditions, especially infections, occasionally results in a more serious prognosis and in some cases is an important contributory factor to the death of a child, e.g. when severe iron-deficiency anaemia is associated with bronchopneumonia, kwashiorkor, tuberculosis, etc. Many workers[4-7] have recorded series of cases of lead poisoning and state that pica is an important aetiological factor in lead poisoning. Lanzkowsky[8] has shown that all the children in his series of cases of pica had iron-deficiency anaemia. Iron-deficiency anaemia therefore, if associated with pica, predisposes to lead poisoning and thus in turn to a fatal outcome or to persistent neurological sequelae. Apart from these overt cases of iron-deficiency anaemia there exists a host of milder cases of iron-deficiency anaemia which remain undiagnosed and cause irritability, lassitude and anorexia and predispose the subject to infection.

Why is it then that iron-deficiency anaemia with its effective and cheap treatment is still prevalent today? Physicians, preoccupied with prescribing vitamin preparations, fail to insist on adequate prophylactic iron—particularly in cases where inadequate iron stores in the infant are anticipated. Adequate prophylactic iron therapy is indicated if the infant is prematurely born, one of a multiple birth or 'later born' compared with a first born, if the infant has lost blood at birth as a result of haemorrhagic disease or faulty ligation of the cord, or if less blood is replaced than is withdrawn in an exchange transfusion, etc. Prophylactic iron therapy is also necessary when maternal iron-deficiency anaemia exists as a result of frequent pregnancies or menorrhagia. The importance of maternal iron deficiency as a predisposing factor in the development of anaemia in infants has for a long time been debatable. The balance of opinion in recent years favours maternal iron depletion as a definite aetiological factor in the development of iron-deficiency anaemia in infancy. It has been shown that while the mean haemoglobin levels of European and Cape Coloured infants at the time of birth are almost identical, the haemoglobin level of the Cape Coloured child at 3 months of age is statistically significantly below that of European infants of the same age.[9] This may be a manifestation of inadequate maternal iron stores in Cape Coloured mothers.

Under these circumstances it is unsafe to rely upon dietary iron alone to prevent the development of iron-deficiency anaemia and it is wise to provide two to three times the usual dose of iron recommended for prophylaxis, i.e. 10-15 mg. of elemental iron daily rather than 5 mg., and therapy should be started at 6-8 weeks of age.

Post-natal factors such as dietary intake of iron, especially in prolonged breast feeding and excessive demand for iron because of rapid growth, are all potent factors in the development of iron-deficiency anaemia.

The presence of milder degrees of iron-deficiency anaemia is by no means always easy to recognize. The insidious onset of the condition, the remarkable ability of the body to adjust itself to a very low haemoglobin level, the vague symptomatology, the association with infection and the notoriously unreliable sign of pallor all combine to confuse the physician. McAlpine *et al.*,[10] in a statistical investigation to determine the value of a clinical assessment of haemoglobin estimation based on facial colour, conjunctival inspection and inspection of the palmar creases, concluded that the clinical assessment of haemoglobin estimations above 60% (taking 14·8 g. as 100%) were so often erroneous that they could not be relied upon in the diagnosis of anaemia, whereas the error was small when the haemoglobin was 50% or less. This is in accordance with Lahey's statement[11] that pallor is rarely seen when the haemoglobin exceeds 7 g.% and only consistently appreciated when the haemoglobin is below 6 g.%. In view of the unreliability of the clinical determination of haemoglobin above 7 g.% it is a sad reflection on our standards of clinical accuracy that we should be satisfied to recognize haemoglobin levels below 7 g.% as the only significant criteria and pass the lesser degrees of anaemia as normal. The wider use of clinical haemoglobinometry is strongly recommended because this is the only way in which the all too frequent and important presence of subclinical anaemia can be recognized.

When iron-deficiency anaemia develops, treatment with an adequate dose of a suitable iron salt is indicated. Ferrous salts have an absorption of 10-15% in the usual doses given to anaemic patients in contrast to an absorption of 1·5-3·0% for iron and ammonium citrate.[12] For this reason ferrous salts in the form of sulphate or gluconate enjoy wide usage. A dose which furnishes 60-75 mg. of elemental iron per day,

given in 3 or 4 divided doses, is satisfactory for patients up to three years of age, after which 2-3 times this amount may be necessary. Four mg. of iron per kg. of body weight per day has been suggested as a minimal effective dose.[11] In addition to the rise in haemoglobin level induced by iron therapy, there are the less tangible effects of iron. Today, few physicians appreciate the marked tonic effect of iron therapy on the appetite, behaviour and sense of well-being of the child.

Until such time as iron is appreciated and made as readily available as certain vitamins, it will continue to be the physician's responsibility to recognize, prevent and treat this all too prevalent deficiency state. This presents a perfect example of the scope of preventive medicine and deserves adequate recognition.

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VERANDERENDE LEWENSPATRONE

Toe ons by 'n vorige geleentheid geskryf het oor *Die ontwortelde mens: 'n probleem van ons tyd*,[1] het ons aangetoon dat die twintigste eeu met sy verbete stryd om lewensruimte, sy onversoenbare politieke ideologieë en sy ontredderende wêreldoorloë vir die geneesheer van die moderne tyd probleme geskep het wat vir sy voorgangers in dié beroep nie bestaan het nie.

Een van die baie redes vir hierdie veranderende probleemgesteldheid van vandag is die feit dat ons leef in 'n tyd waarin daar dwarsoor die wêreld groot ekonomiese en kulturele verskuiwings aan die gang is wat soms hele rasse en volke insluit. Verskillende etniese groepe leef op alle grade van ontwikkeling naby mekaar en langs mekaar. Terselfdertyd is daar 'n wegkrimping van grense en skeidslyne—omstandighede wat onder andere die gevolg is van snel-veranderende metodes van vervoer en verbinding in die moderne wêreld.

Die fundamentele vraag waarvoor ons almal dus vroeër of later te staan moet kom, is die vraag of daar wel universele kulturele en gemeenskapspatrone bestaan wat aanleiding gee tot òf gesonde en behoudende lewenswyses òf ongesonde en morbiede patrone van lewe. Vir die dokter wat daagliks met mense van alle soorte op alle vlakke te doen het, is hierdie vrae van oorwegende belang—soos hulle ook vir die politikus is wat die lot van miljoene mense in die holte van sy hand hou.

Die uitdaging aan almal wat met mense moet werk—en dus veral ook aan ons as dokters—is om so 'n objektiewe studie as moontlik te maak van menslike verhoudings tussen indiwidue en groepe, teen 'n agtergrond van uiteenlopende persoonlike, maatskaplike, ekonomiese, kulturele en politieke faktore.

Omdat ons uit die aard van die saak nie navorsingswerk van hierdie soort self kan onderneem nie, moet ons ons wend tot die werk van ander ondersoekers op hierdie gebied. Nou het daar onlangs 'n boek verskyn, *African Interlude*[2], wat geskryf is deur dr. J. F. Holleman, 'n bekende antropoloog, wat ses jaar van sy lewe tussen die Mashona-stamme van Suid-Rhodesië deurgebring het. Sy bevindinge, wat met groot sorg en besondere deursettingsvermoë vergader is, word hier weergegee in 'n interessante verhaal, verryk met belangrike inligting aangaande die stam- en huweliksgewoontes, verwantskapstelsels, maatskaplike en politieke organisasie, grondregte, kontrakregte, hofgewoontes, ens. by die stamme onder wie hy gewerk het. Die persoonlike ervarings van die skrywer is op interessante wyse verbeeld saam met die verhaal van sy navorsingswerk.

In die wêreld van vandag is daar orals mense wat baie sterk menings nahou ten opsigte van die probleme van kulturele verskuiwings van die moderne tyd. Dikwels is hierdie menings op ongenoegsame gronde gebou. 'n Boek soos hierdie, waarin feite aan ons voorgestel word sonder bevooroordeelde kommentaar, is dus van die grootste waarde.

Hierdie boek is die soort werk wat ons sal help in ons eie strewe na objektiewe kennis op grond waarvan ons dan ons eie voornemens kan uitvoer: Eerstens om ou patrone van maatskaplike en kulturele lewe wat verander en groei, of agteruitgaan en verdwyn, te bestudeer; en tweedens om die uitwerking van veranderende lewensomstandighede op die geestelike en liggaamlike welsyn van persone en groepe te bepaal.

1. Van die Redaksie (1958): S. Afr. T. Geneesk., 32, 700.
2. Holleman, J. F. (1958): *African Interlude*. Kaapstad: Nasionale Boekhandel Beperk.

BREAST CANCER CAMPAIGN

The Cape Western Branch of the National Cancer Association of South Africa is planning an intensive education campaign among women in the Cape Peninsula to emphasize the need for early diagnosis and treatment in breast cancer. The Cancer Association is anxious to solicit the wholehearted support of the medical profession to ensure that doctors, who may be consulted as a result of this campaign, will be aware of it and of the effect it may have had in bringing patients to them.

The Cape Western Branch of the National Cancer Association of South Africa has decided to ask the Medical Association to arrange a preview of a film reserved for the medical profession entitled 'Breast Cancer—The Problem of Early Diagnosis'. At the same time it is felt that it would be useful for the medical profession to see the public education film 'Breast Self-Examination'. This film will only be shown to the public when a medical lecturer is available to lecture and answer questions on the subject.

It has been suggested that these two films be shown to doctors at a meeting in the Physiology Lecture Theatre, Medical School, Observatory, Cape, on 27 February 1959. This film was also shown to doctors attending the University of Cape Town General Practitioners Revision Week. Adequate time will be given for members of the medical profession to express their views before the National Cancer Association show the film 'Breast Self-Examination' to the public.

Additional information regarding these films may be obtained from the Education Sub-Committee, Cape Western Branch of the National Cancer Association, P.O. Box 2390, Cape Town.

VENTRICULAR-SEPTAL DEFECT WITH AORTIC INCOMPETENCE

MAURICE NELLEN, M.D. (CAPE TOWN), M.R.C.P. (LOND. AND EDIN.), VELVA SCHRIRE, PH.D., M.B., CH.B. (CAPE TOWN), M.R.C.P. (LOND. AND EDIN.), and LOUIS VOGELPOEL, M.B., CH.B. (CAPE TOWN), M.R.C.P. (LOND.)

From the Cardiac Clinic, Groote Schuur Hospital, and Department of Medicine, University of Cape Town

We are describing the clinical, cardiac catheterization and necropsy findings in a boy aged 15, with ventricular septal defect and aortic incompetence. The clinical findings are typical of this rare combination of defects. We also review the previously reported cases.

CASE REPORT

P.G., a European male aged 15, was first seen in September 1956 with the following history. During his mother's pregnancy no illness of note had occurred. At the age of 2 months attention was first drawn to his heart by the discovery of a cardiac murmur. Development during infancy and early childhood had been normal, though he was never robust physically and always complained of easy fatigue and dyspnoea, with palpitations on effort. Cyanosis and squatting were never present. At the age of 6 he was taken to Boston, where he was examined by Dr. Robert E. Gross, who found a loud blowing systolic murmur, most intense over the central part of the chest and considered he had a ventricular septal defect.

For 2 years before examination he had been complaining of a sharp lower chest pain on the right side, stabbing in nature, occurring with effort and relieved by rest. His general condition had deteriorated steadily and he had become noticeably more breathless on effort.

He was now a thin, normally developed youth. Cyanosis and clubbing of the fingers was absent. There was no abnormality of his palate, fingers or toes. The jugular venous pulsations and pressure appeared normal. His peripheral pulses were collapsing in type with a Corrigan pulse in the neck and bounding femorals. The blood pressure was 140/60-40 mm. Hg.

There was a slight left-sided praecordial bulge with apical pulsations in the 6th left intercostal space 4 inches from the mid-sternal line. The apex was formed by an overfilled, hyperdynamic left ventricle. A rough systolic thrill was present, felt maximally at the 3rd left intercostal space parasternally. A harsh pan-systolic murmur (grade 5/6) was present, of maximal intensity in the 3rd and 4th left intercostal spaces, close to the sternum. This murmur was suggestive of a ventricular septal defect with a left-to-right shunt. At the base the murmur was different, being shorter, with a mid-systolic crescendo and ending before the aortic second sound. The characteristics were those of an aortic ejection systolic murmur.[32] A long, loud early diastolic murmur was present, best heard parasternally at the 2nd and 3rd left intercostal spaces. Splitting of the second sound could not be detected. The above findings were confirmed on phonocardiography (Fig. 1). At no time were the murmurs thought to be fistulous. In the pulmonary area the findings were clearly those of a to-and-fro murmur. However, at the 4th left space, although the murmurs were 'continuous' in the sense that systolic and diastolic murmurs fused at the second sound, they could be distinguished as murmurs of separate origin.

The electrocardiogram (Fig. 2) showed the pattern of left ventricular hypertrophy. The chest film (Fig. 3) showed considerable enlargement of the heart affecting chiefly the left ventricle, with marked enlargement of the pulmonary arteries and pulmonary plethora. Screening confirmed the general configuration of the heart and a left-to-right shunt was suspected in view of the dilated, vigorously pulsating pulmonary arteries with pulmonary plethora.

The diagnosis of ventricular septal defect with aortic incompetence was made and, as the question of cardiac surgery was seriously considered, cardiac catheterization was performed and the following data obtained: The mean pressure in the right auricle was + 2 mm. Hg and the venous pulse waves were normal. The pressures in the right ventricle varied from 55/0 low down to 35/0 high up in this chamber (Fig. 4). The pulmonary-artery pressure was 38/18 and the mean 'wedge' pressure 13. The systemic pressure at this time was 140/40. Samples from the chambers of the heart showed an oxygen saturation in the right atrium and venae cavae of 68% (Haldane), a right ventricular and pulmonary-artery saturation of 86%, with a brachial-artery saturation of 97%. The systemic flow (calculated on the Fick principle) was 4·8 litres per minute, with a pulmonary flow of 13·2 litres per minute and a pulmonary resistance of 1 unit or 80 dynes sec/cm^5. The catheter findings were regarded as confirming a large ventricular septal defect, with a considerable left-to-right shunt at the ventricular level. The reason for the difference in pressure between the upper and lower parts of the right ventricle was not immediately apparent and is discussed later.

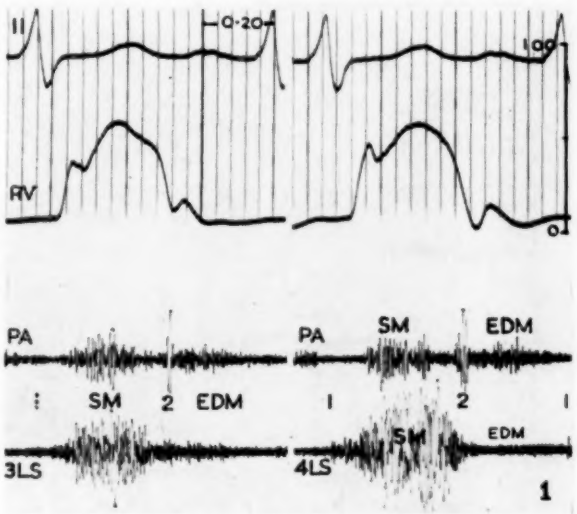

Fig. 1. Phonocardiograms recorded simultaneously with ECG and right ventricular pressure tracing (RV). In the pulmonary area (PA) there is an ejection systolic murmur (SM) with a mid-systolic crescendo, and an early diastolic murmur (EDM) with a crescendo-decrescendo configuration during diastole. The features are those of a to-and-fro murmur and not a fistulous murmur. In the 3rd left interspace (3LS), where the second heart sound is soft, the systolic murmur appears continuous with the diastolic murmur, but the mid-systolic crescendo distinguishes the systolic murmur from the late crescendo found in patent ductus arteriosus. In the 4th left interspace (4LS) a different systolic murmur is recorded. It is much louder, has a crescendo later in systole, and extends into the second sound. It is the murmur produced by a left-to-right shunt through a ventricular septal defect. The early diastolic murmur at this site is much softer than the systolic murmur and although continuous with the systolic murmur did not give the auscultatory impression of a fistulous murmur.

He was discharged until arrangements for surgery could be made. However, urgent readmission was necessary in February 1957 because of severe deterioration in his condition. Breathlessness had rapidly increased until he was orthopnoeic. Palpitations were very troublesome and severe heaving of the chest became noticeable to the patient. Abdominal discomfort, loss of appetite and vomiting followed. For 1 week before admission swelling of the legs and face occurred.

The boy was now found to be extremely ill, semi-stuporose,

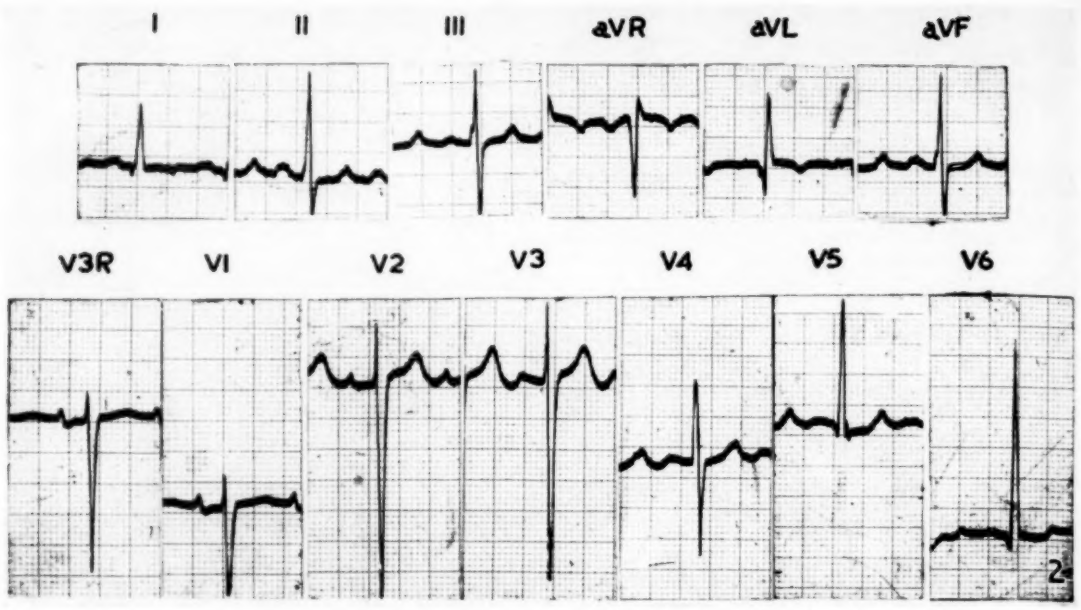

Fig. 2. Electrocardiogram, showing left ventricular hypertrophy.

and with Cheyne-Stokes breathing. Marked tachycardia was present, but no cyanosis. There was marked oedema of the legs, thighs and sacrum, with ascites. The jugular veins were so distended that the pressure could not be assessed clinically, and the liver was 3 fingers below the costal margin, firm and tender. The murmurs were essentially unchanged. The blood pressure was 150/40-0. The electrocardiogram remained unchanged. The X-ray now showed considerable increase in the cardiac size, especially involving the ventricles and pulmonary arteries (Fig. 5). Congestion of the lungs was marked.

Treatment was commenced with digitalis, mercurials and aminophyllin. Penicillin and streptomycin was administered for suspected subacute bacterial endocarditis, despite negative blood cultures. His general condition deteriorated, however, without relief of his acute dyspnoea and he died 4 days after admission.

Necropsy Findings: The findings were those of ventricular septal defect with aortic incompetence. The heart was grossly enlarged (weight 790 g.). The left ventricular wall was 1·5 cm. thick and measured 12·5 cm. in length from the aortic region to the apex. Beneath the medial aortic cusp a ventricular septal defect was present, measuring 2·5 cm. by 1 cm. The lower edge was semilunar in shape and a thickened ridge of endocardium demarcated the boundary (Fig. 6).

The upper limit of the defect on its left ventricular aspect consisted of two of the aortic valve cusps. These cusps were freely mobile, the surfaces being irregularly thickened. The third cusp was less severely thickened. The medial aortic cusp overhung the septal defect and was markedly thickened and displaced medially into the defect; thus the aortic cusps could not be approximated in diastole, resulting in the clinical aortic incompetence.

The posterior papillary muscle of the left ventricle was considerably hypertrophied. A few small linear areas of endocardial thickening were present, presumably the result of aortic incompetence. The sinuses of Valsalva were normal. The right ventricle and auricle were dilated, the former measuring 0·5 cm. thick, with papillary muscles as large as those normally found on the left side. Viewed from the right ventricular cavity (Fig. 7), the defect was directly beneath the medial leaflet of the tricuspid valve. The ventricular septum below the defect bulged convexly towards the right ventricular chamber, hiding the defect from view when seen from below. From the site of the defect and its relation to the tricuspid valve the jet of blood from the left ventricle to the right ventricle may well have impinged on the under surface of the medial leaflet of this valve.

DISCUSSION

The diagnosis of ventricular septal defect was made in this case on the basis of a loud pan-systolic parasternal murmur associated with left ventricular enlargement and a left-to-right intracardiac shunt on screening. The presence of bounding carotids and a collapsing pulse in association with an ejection systolic and early diastolic murmur at the base suggested aortic incompetence. The combination of a ventricular septal defect with aortic incompetence would account for the marked clinical electrocardiographic and radiological evidence of left ventricular enlargement.

The same haemodynamic findings are encountered in a large patent ductus or aortico-pulmonary septal defect. However, in these conditions the murmurs are truly continuous with a late systolic crescendo[33] and decrescendo diastolic murmur and are best heard in the pulmonary area. In our case, the murmurs at the pulmonary area were clearly not continuous, being to-and-fro (Fig. 1.) However, at the 4th space, although in a sense 'continuous', the crescendo of the pan-systolic murmur was in late systole and the diastolic murmur had a crescendo-decrescendo configuration as found in aortic incompetence[42] (Fig. 1). In a ruptured sinus of Valsalva with an aortic-right ventricular shunt, the murmurs are sometimes loudest at the 4th space, simulating very closely ventricular septal defect with aortic incompetence. However, in our case, the history of a loud, low parasternal systolic murmur without any diastolic murmur during childhood strongly favoured the presence of a ventricular septal defect rather than an unruptured aneurysm.

TABLE I: 46 CASES OF VENTRICULAR SEPTAL DEFECT WITH AORTIC INCOMPETENCE, WITH AVAILABLE CLINICAL AND PATHOLOGICAL DATA

(a)	X-ray (b)	ECG	Blood Pres.	Murmur	Age (years) First seen	Age (years) Death	Pathology	Clin. Diagnosis
2	H III	abnormal	130/20	to-and-fro	7	15	VSD 1·5 × 1·2 cm. R aort. cusp protruded into RV occluding VSD. Fused with ant. pulm. cusp. Wt 575 g.	PDA or AI
4							VSD + AI	
3	LA II LV II H IV+ PA II	LV +	110/40	S and D 'continuous'	2/12	3	VSD + R aort. cusp herniation through VSD and adherent to pulm. valve. Wt 180 g.	PDA + VSD
5	H IV PA I Aol	LV+	110/30	At age 6 only syst. At age 9 S and D and thrills.	8/12	9	Ballooned ant. aort. cusp completely hiding the high VSD. Defect 2 × 1·2 cm. LV + + +. Aorta overrides septal defect. Wt 500 g.	? aort.-pulm. septal defect
	RV I LV III	rs-RS in VI, RV+	100/60	left sternal edge S and D		8	RV wall 9 mm. thick. VSD 20 mm. across. LV 11 mm. thick. Aort. valves override VSD. Wt 370 g. Normal for age 115 g.	VSD + pulm. hypertension
8							VSD + AI	
12	H IV+ heart H IV+					15	VSD + AI	VSD + AI
15	PA II LV II RV II	LV + + RV + +	130/40-0	to-and-fro S and D	1/12	6 (died at op.)	'Hypertrophy and inf. displacement of the non-coronary cusp of the aort. valve.' VSD in membranous part, 1 cm. across. Endocarditis due to *Str. viridans*. Wt 310 g.	VSD + AI PDA
16			84/0	S and D		14	VSD 1 cm. diam. Almost occluded by pouch-like dilatation of ant. aort. cusp protruding through VSD. Wt 150 g.	PDA
20	H IV LV I RV I PA I	LV+	130/0	S and D	20	21 died suddenly	RV 11 mm. thick, LV 17 mm. thick. High VSD 23 mm. long. Aort. cusp over the defect was as large as the other 2 together and was fenestrated, leading to insufficiency. Death from heart failure	PDA
23	(12 cases)			Murmurs not continuous more properly 'to-and-fro'. Diastolic murmurs may change from time to time. Low diastolic pressure and marked aortic pulsations				
26	H IV	LV + + RV + +	145/80-50	S. Aort. incompetent murmur first heard age 20			VSD + roughening and calcification of R aort. cusp and sac-like dilatation of cusp. Slightly dextroposed aorta. Wt 675 g.	PDA
30							High VSD. Aort. cusp drawn down by fibrous band towards defect, rendering valve incompetent.	
31	(5 cases) H large		'high pulse pressures'	Loud aort. D		18	2 cases died and both had high VSD to which a cusp of the aort. valve was tethered by a fibrous band. L case cyanosed before death	
33	LV I PA II	LV+	180/60	'continuous' holosystolic	34			PDA + VSD
37	H III PA III LV II RV II	RV + LV +		loud S and D	13	13 (died at op.)	Septal defect 2·5 cm. long. Some dextroposition of aorta. LV + + and RV + +. Aort. cusps deformed. Wt 670 g.	PDA
39	H II		112/0	to-and-fro. Aort. D.	7	7	Died from staph. endocarditis. Aorta partially overlay the VSD. Right aort. cusp thickened; its line of closure markedly retracted and lay at a lower level than the other cusps leading to AI. Defect 1·75 cm. in diam.	? VSD ? 1. subaort. stenosis ? 2 bicuspid aort. valves ? 3 congen. aneurysmal dilatation of sinus of Valsalva
43 (c)	H III LV I RV I PA I	RBBB	160/0	to-and-fro	37	56	VSD 1·5 × 5 cm., high in membranous septum, immediately beneath right cusp of aort. valves. Also aneurysm of sinus of Valsalva, opening into RV beneath left cusp of pulm. valve. R. aort. cusp thickened. AI. Wt 960 g.	PDA
	LV III RV III PA III LV III RV III PA III	LV+ Auric. fibril.	160/60	to-and-fro	13	33	Most enlargement in RV. Staph. endocarditis in mitr. and aort. valves. High membranous VSD, 2·5 cm. diam. R aort. cusp immediately above was medially displaced and inferiorly thickened, preventing closure of aort. valve	
45	LV II RV I	LV + +	110/50	S and D	1	21	VSD 2 cm. in diam. Ant. aort. cusp partly prolapsed into defect and much thickened at its edge. Extensive subendocardial fibrosis at base of valve	VSD + AI
46	LV II PA II	LV+	140/50	S and D	17			VSD + AI PDA
	LV II PA II	LV+	125/45	S and D	24			PDA
	RV II LV II PA II	LV+	180/60	S and D	Infancy			VSD + AI
48								VSD
47								VSD + AI

Abbreviations: H = heart. RV = right ventricle. LV = left ventricle. PA = pulmonary artery. Ao = aorta. VSD = ventricular septal defect. PDA = patent ductus arteriosus. AI = aortic incompetence. RBBB = right bundle-branch block. S = systolic. D = diastolic. Grade of enlargement: mild = I, moderate = II, severe = III, gross = IV.

(*a*) This column shows who reported the cases (numerals refer to list of references on page 96)

(*b*) The roman numerals refer to grades of enlargement.

(*c*) Six cases, of which details of only 4 are set out.

The findings at cardiac catheterization indicated a left-to-right shunt at the ventricular level. The absence of pulmonary hypertension was confirmed, and the increased saturation in the right ventricle could not be attributed to pulmonary incompetence. A ruptured sinus of Valsalva was not completely excluded on the catheter findings.

The natural history of this case is of great interest. At the age of 6 a loud systolic murmur only was present, presumably caused by the ventricular septal defect. With the passage of time the aortic incompetence developed and gradually became a feature of the boy's condition. In fact, the ultimate development of gross aortic incompetence must be regarded as the major contributory cause of death. The later development of aortic incompetence has also been reported by Baylis *et al.*[5] and Hurst and Schemm.[26]

About 10% of ventricular septal defects are openings due to imperfections in the formation of the muscular septum, which may be located in various parts of the septum, whereas 90% of the isolated ventricular septal defects and virtually all such defects occurring in combined malformations of the heart are located in the region of the membranous septum.[35] The term 'membranous' is inadequate, because the defect also involves the large zone of muscular tissue adjacent to the membranous septum. When the defect is large it involves the septal tissue immediately below the annulus of the aortic valve. The aortic leaflets are thus seen to overhang the defect.[27] The aortic cusp which lies above the septal defect is often abnormally large and scarred.[39]

The defect, when viewed from the right ventricle, involves the right ventricular outflow tract fairly low down below the crista supraventriculars[41, 27] and is well separated from the pulmonary valves. When viewed from the left ventricular aspect, on the other hand, the defect is seen to lie immediately beneath the aortic valve cusps[27] and it can readily be understood how the aortic valve cusp overlying the defect may be affected by a stream of blood flowing from the left to the right ventricle.

The case described here represents such a defect and, in addition, the aortic cusp overlying the defect was slightly lower, thickened and scarred, and seemed to hang as it were in mid-air, resulting in aortic incompetence. It seems that the changes found in this cusp may be slowly acquired as a result of trauma or motion. It is also possible for the cusp to be dragged downwards by the Venturi effect of a jet of blood passing below it from the left to the right ventricle through the defect.

In some cases[3, 45, 2] the right aortic cusp may actually protrude through the ventricular septal defect into the right ventricle, partially occluding the defect. In 2 cases[3, 2] the protruded valve adhered to the pulmonary valve. In a case described by Hurst and Schemm[26] the right aortic cusp was calcified. An associated aneurysm of the sinus of Valsalva opened into the right ventricle. Laubry *et al.*[31] described a case in which the right aortic cusp was tethered to the septal defect by a fibrous band. In a case described by Espino-Vela and Mata[20] the right aortic cusp was twice as large as either of the other two aortic cusps and was fenestrated, resulting in incompetence.

Ventricular septal defect with aortic incompetence is not common and, according to Collins *et al.*,[46] was first described by Breccia[47] in 1906 and then by Laubry and Pezzi[30] in 1921. Wood[44] states that the frequency of this association in one series was 2%. As far as we are aware from the literature (Table I) our case is the 47th published case and the 8th to be catheterized.[15] A study of the reported cases (Table I) reveals the remarkable uniformity both of the clinical and pathological findings. Most cases were seriously handicapped. All had a high systemic pulse pressure with evidence of aortic incompetence and marked carotid pulsations.

Although the murmurs are often described as 'continuous', it is likely from the descriptions and from the phonocardiograms published that the systolic murmur of ventricular

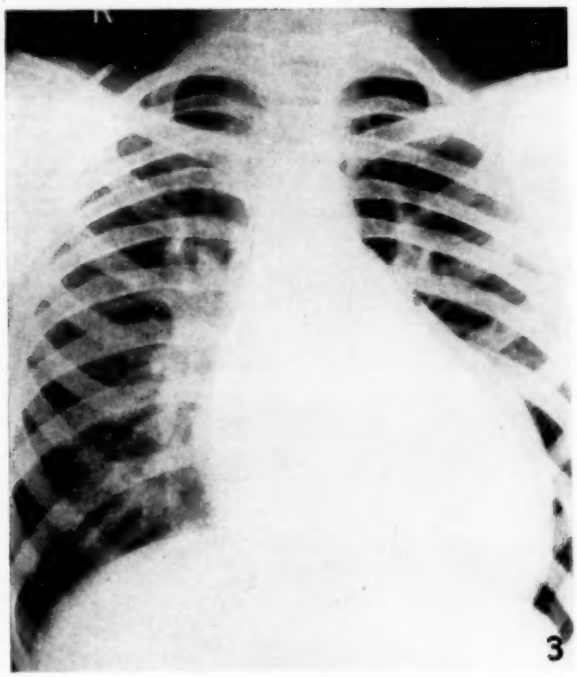

Fig. 3. X-ray of heart, 12 October 1956, showing dilated pulmonary arteries, marked pulmonary plethora, and enlargement of right and left ventricles.

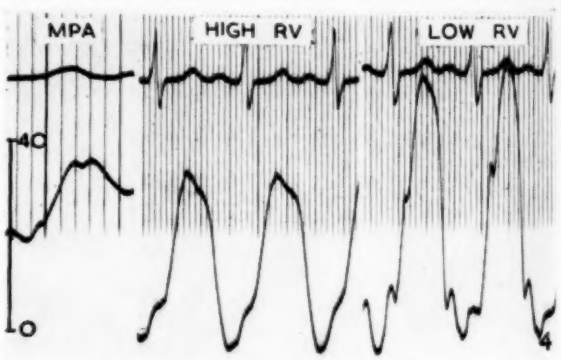

Fig. 4. Cardiac catheterization findings, showing pressure tracings in main pulmonary artery and high and low right ventricle. Note similar systolic pressures of 35 mm. Hg in main pulmonary artery and high right ventricle with a low ventricular systolic pressure of 55 mm. Hg.

Fig. 5. X-ray of chest 16 February 1957, showing gross cardiomegaly and extreme pulmonary plethora and dilatation of all chambers of the heart.

Fig. 7. The specimen shows the cavity and outflow tract of right ventricle. Probe 1 is in ventricular septal defect. Probe 2 is on muscular septum and 3 is on hypertrophied outflow tract below pulmonary valves.

septal defect and the early diastolic murmur of aortic incompetence are present, as in our case.

The electrocardiograms, where reported, showed the signs of severe left ventricular hypertrophy, as found in our case.

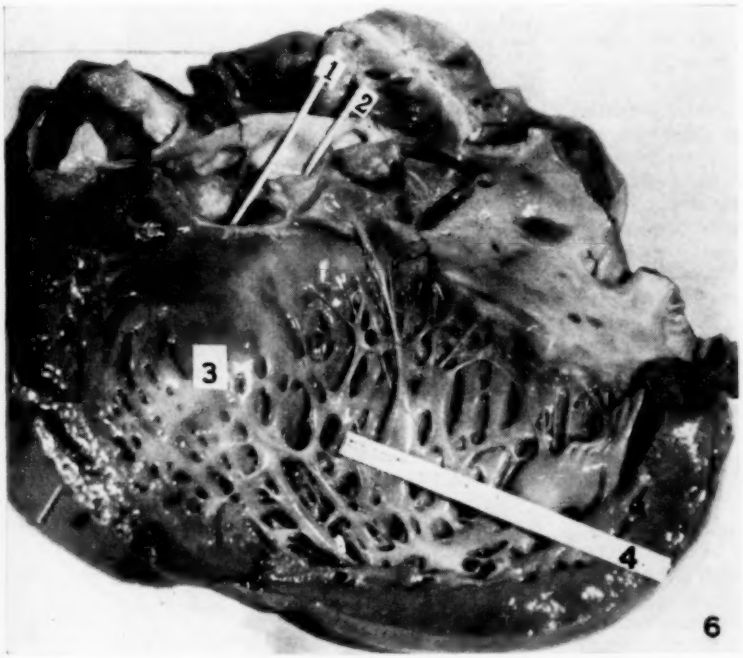

Fig. 6. Heart viewed from left ventricular cavity. Probe 1 is through the ventricular septal defect, just below the hypertrophied, thickened and prolapsed medial aortic cusp. Probe 2 is in the middle aortic cusp. No. 3 is on interventricular septal muscle. No. 4 is on rule showing thickness of left ventricular wall of 15 mm.

Radiograms of the heart in most of the reported cases have shown gross cardiomegaly. The left ventricle is the most severely involved chamber, because of the aortic incompetence, but the associated radiological signs of a ventricular septal defect such as pulmonary plethora and right ventricular enlargement also occur.

The association of the two defects results in severe strain on the left ventricle and carries with it a serious prognosis.

Death in heart failure is the rule, but 3 cases have been described where the cause of death was infective endocarditis,[43, 15, 39] 2 from staphylococcal endocarditis and 1 from *Streptococcus viridans* infection. In 2 of the cases, the endocarditis involved the deformed aortic cusp and the mitral valve, the ventricular defect escaping. None of the 41 previously reported cases were cyanosed except for one,[31] and this patient was cyanosed only while in severe congestive cardiac failure.

The pathogenesis of aortic incompetence in cases with ventricular septal defect is worthy of speculation. It would appear to depend on the presence of a large left-to-right shunt through a septal defect of some size just below the valve. In support of this the

rarity of aortic incompetence in cases of equally large ventricular septal defects where there is no left-to-right shunt, and where there are balanced ventricular pressures either from severe pulmonary hypertension (Eisenmenger's complex) or severe pulmonary stenosis (Fallot's tetralogy).

Although it has been stated that aortic incompetence complicates Eisenmenger's complex,[38, 9] which is by definition 'pulmonary hypertension with reversed interventricular shunt, the cyanosis being accounted for entirely by the right-to-left shunt through the ventricular septal defect,' [36, 44, 18, 12] we have been unable to find an acceptable published case of Eisenmenger's complex with pathological proof of the co-existence of aortic incompetence. Of the 31 cases of Eisenmenger's complex collected from the literature up to 1951 with pathological proof,[36] none had evidence of aortic cusp deformity at necropsy. Although an early diastolic murmur is described in many of these cases, the clinical and pathological evidence seems to point to pulmonary incompetence as the cause. Wood[44] mentions that a Graham Steell murmur due to functional pulmonary incompetence was heard in 2/3 of all his cases of Eisenmenger's complex. No mention is made of aortic incompetence. Early diastolic murmurs were found in 6 of our 9 catheterized cases of Eisenmenger's complex and these, on good clinical grounds, were regarded as pulmonary and not aortic in origin. The differentiation between the early diastolic murmur of aortic incompetence and pulmonary incompetence is sometimes very difficult and often depends on the associated findings. Thus, the presence of a collapsing pulse in the systemic arteries indicates that the early diastolic murmur is aortic in origin, whereas in pulmonary incompetence the systemic pulse is normal or small. Furthermore, there are usually signs of severe pulmonary hypertension when the early diastolic murmur is pulmonary in origin.

In a further 83 cases of Eisenmenger's complex published since 1951 with either catheter or necropsy proof reviewed by us, we were unable to find any example of aortic incompetence.[6, 17, 13, 14, 12, 22, 25, 26, 29, 34, 44] It would therefore appear from a review of the literature that aortic incompetence is exceptional in the Eisenmenger's complex if it ever occurs.

In Fallot's tetralogy aortic incompetence is also most uncommon. We have encountered one case of trivial aortic incompetence in our series of 51 proven cases of Fallot's tetralogy and 1 case of trivial pulmonary incompetence in a mild tetralogy. In the tetralogy the differentiation is readily made because an early diastolic murmur following immediately after the second sound is aortic in origin, where as a pulmonary incompetent murmur commences well after the aortic second sound. This is due to the fact that pulmonary valve closure is always much delayed in that condition.[40]

As the combination of aortic incompetence and ventricular septal defect appears to occur most of all in cases with a large left-to-right shunt and is rare, if it occurs at all, where the ventricular pressures are balanced with a reversal of the shunt, it is not unreasonable to attribute the development of the valve lesion to the left-to-right shunt. Such a shunt may tend to displace and deform a vulnerably placed aortic cusp and in time result in its incompetence.

The pressure differences found in the right ventricle are of some interest. Thus a consistently higher pressure was recorded in the body of the right ventricle compared with that in the outflow tract (Fig. 4). This is presumably due to some obstruction to flow in the outflow tract and not to a Venturi effect, for the pressures well down in the pulmonary arteries were also significantly lower than in the body of the right ventricle. At necropsy, while no organic stricture could be demonstrated, marked hypertrophy of the outflow tract was present. This may account for the small pressure gradient in the ventricle. That hypertrophy of the outflow tract may result in subvalvular obstruction is now well recognized in both ventricles,[10,11,48] Interesting supporting evidence for this view comes from two recent reports. Gasul *et al*,[21] catheterized 2 patients with ventricular septal defects in early infancy, at which time the pressures in the right ventricle were all identical. Four years later, re-catheterization in both showed the development of a definite gradient with markedly higher pressures in the body of the right ventricle. This type of obstruction is apparently reversible, as Engle *et al.*[19] have shown in pulmonary stenosis. Three patients were observed to have a right ventricular pressure of over 100 mm. Hg after pulmonary valvotomy performed under hypothermia. The obstruction that remained was localized to the outflow tract of the ventricle. Cardiac catheterization many months later showed pressures within normal limits with only slight or no transvalvular gradients. Nine of 21 cases of ventricular septal defect operated on by Cleland *et al.*[48] had infundibular obstruction and similar intraventricular pressure gradients as described in our case.

SUMMARY

1. A case of ventricular septal defect with aortic incompetence from cusp deformity is described.

2. The clinical, electrocardiographic and radiological findings are described and related to the findings on cardiac catheterization. The unusual pressure findings in the right ventricular chamber are discussed.

3. The necropsy findings are reported.

4. The literature is reviewed.

ADDENDUM

Since this manuscript was accepted for publication 8 more cases have been reported, [49,50,51] making a total of 54 reported cases. Three of the cases were confirmed by autopsy, 2 were proved by thoracotomy, 6 were catheterized and in 2 a retrograde aortogram aided the diagnosis. The authors stress the diagnostic difficulties and mention cardiac catheterization, retrograde aortography and dye-dilution studies as aids in diagnosis.

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BASAL-CELL CARCINOGENESIS

CASE REPORTS ILLUSTRATING A TWO-STAGE MECHANISM

T. SCHRIRE, M.A., M.B., CH.B. (CAPE TOWN), F.R.C.S. and ROSSALL SEALY, M.A. (CANTAB.), M.MED. (RAD. T.) (CAPE TOWN)

Combined Clinic, Groote Schuur Hospital and Departments of Surgery and Radiotherapy, University of Cape Town

The experiments of Deelman *et al.*[1] have, for many years, been accepted as supplying a reasonable basis for explaining some of the actions of carcinogens and the genesis of cancer. Deelman painted the skin of mice with a tarry preparation, in itself not a carcinogen, and months later, with the skin apparently returned to normal, made incisions in the areas that had been tarred. Neoplasms, admittedly papillomata, appeared in the lines of the surgical incisions and a large body of literature has grown up in explanation of these findings. Soon after this similar experiences were being advanced to explain malignant lesions[2] and experimental production of many of these has been explained on the basis of Deelman's work.

It is believed that the original tarring caused an initiatory process which was slow-acting and irreversible. Whilst the cells affected showed no chemical or histological deviation from the normal, something had happened to sensitize them so that the second type of stimulus—the promoting stimulus—which need not necessarily be chemical, caused these cells, and these cells only, to become neoplastic.

Considering a large series of facial and lip cancers[3] it seemed to us that Deelman's explanation of the genesis of neoplasms as a multi-stage mechanism could probably be applied to these tumours. De Kock[4] had noted the development of actinic cancer in white rats exposed for some months to sunlight occurring on spots which could be easily scratched or otherwise traumatized, and reports of this type are not rare. Nearly all the skin cancers that developed were, however, squamous epitheliomas. The experimental production of basal-cell carcinoma is a very rare phenomenon. A squamous epithelioma is the commonest cancer produced and seems to be a 'normal' neoplastic response to irritation. Chimney sweep cancer, Kangri cancer, cancers which develop on old standing fistulae or sinuses, or neoplasms which develop on areas over-exposed to irradiation, have almost invariably been squamous epitheliomas.

Cancers which develop on old burns (Marjolin's ulcer) form an interesting group. They are usually epitheliomas but Treves and Pack[5] reported a series of 21 squamous epitheliomas and 7 basal-cell carcinomas in a series of burns and noted that the basal-cell carcinomas usually occurred in superficial burns where the hair follicles and sweat glands had been spared. This agrees very well with our conception that basal-cell carcinomas originate in the sebaceous glands since these had probably been spared in such superficial burns together with the sweat glands and hair follicles already noted. Haagensen[6] found 3 cases of basal-cell carcinoma which developed in small burns of the face. These may have been the neoplastic responses to a promoting stimulus to a previously sensitized area, the smallness of the burn probably allowing the sebaceous glands in the region to be likewise spared.

CASE REPORTS

Case 1

A European male, aged 67, born in England, came to South Africa over 25 years ago. He was employed in the Post Office as a linesman and worked out of doors for 25 years. The patient had blue eyes and grey (once fair) hair. His cheeks were ruddy and showed no trace of solar keratosis. However, the dorsum of his hands were rough and keratotic but no areas were sufficiently heaped up to require biopsy.

Six months ago a dog attacked him and he fell and sustained cuts on both his cheeks and his nose by falling against some bricks and tin cans which happened to be lying about. The wounds on his face healed and 6 months later he presented with 3 lesions exactly in the scars of the wound, *viz.* on the bridge of the nose, on the right cheek in front of the ear and on the chin. Snips from all 3 areas were taken for biopsy and the pathological report reads as follows:

(1) 'The biopsy from the *nose* shows a squamous papillomatous lesion with a pronounced inflammatory cell infiltration in the underlying corium. There is some atypicality of the epithelium and the lesion is probably best regarded as a form of *senile keratosis*.

(2) The biopsy from the *chin* shows 2 *small epidermoid*

cysts and a chronic inflammatory cell infiltration in the corium. The overlying epidermis shows slight hyperkeratosis and occasional minute foci of *basal-cell proliferation* but no evidence of frank malignancy.

(3) The biopsy from the *right cheek* shows infiltration of the subepidermal tissue by foci of *basal-cell carcinoma.*

We did not think that the first and second reports were sufficiently definite and the biopsy was repeated on these 2 areas. The report read as follows:

(1) 'The snip from the *chin* shows hyperkeratosis and intra-epidermal pustule formation. The corium shows a pronounced chronic inflammatory reaction and contains a tiny epidermoid cyst. Acanthosis is pronounced and towards one end of the snip there is elongation and apparent downgrowth of rete pegs. Several sections were examined and in one area some of the more deeply situated epithelial cells show sufficient atypicality to *make one suspect early squamous carcinomatous change*. Proof of this is, however, lacking.

(2) The biopsy from the *nose* also shows hyperkeratosis, intra-epidermal pustule formation and pronounced chronic inflammatory changes in the corium. The evidence of epithelial atypicality and downgrowth is, however, more clear-cut than in specimen (1) and we feel justified in labelling this as an *early squamous carcinoma.*'

In accordance with our accepted plan of treatment, the patient was given radiotherapy to all 3 lesions—all healed uneventfully.

Case 2

A European male, then aged 71, who had been a gardener for 54 years, attended hospital in 1955 complaining of a sore on the right side of the chin which had been present for 6 weeks. Biopsy showed the lesion to be a rodent ulcer, which healed after a course of superficial X-ray therapy. No further lesions developed until 1958 when, whilst shaving, the patient cut his right cheek. The area bled and scabbed but never healed. When seen at hospital 6 weeks later there was a typical crusted rodent ulcer (confirmed by biopsy) in the exact position of the incision. At this time a hyperkeratosis was noted on the right temple and the whole skin of the face showed a fine dry scaliness and loss of elasticity—appearances typical of solar keratosis.

DISCUSSION

We have here clear histories of long exposure to sunlight in persons of typical diathesis, who developed visible solar keratosis of a minor degree only. We presume that as a result of the long exposure the preliminary initiatory process of carcinogenesis had taken place and that many years later a series of simple traumata acted as promoting stimuli and caused the development of squamous epithelioma and of basal-cell carcinoma in 2 separate and distinct areas in the first case, and of basal-cell carcinoma in the second case.

The cases illustrate 2 points: Firstly that the skin can be sensitized without the clinical development of frank solar keratosis, and secondly the variability of the neoplastic response. In case 1 there developed in one place squamous epithelioma, in another acanthosis and in the third site frank basal-cell carcinoma. In case 2 a straightforward basal-cell carcinoma was found. The development of this last type of neoplastic response is very rare and we record it for the first time in our experience. The small epidermoid cysts found in case 1 are not unexpected in view of the nature of the injury and the scratches from the bricks; they are probably inclusion dermoids. Their presence tends to incriminate the injury as being the promoting process.

We are particularly pleased to be able to report these cases because they bear out, almost to the letter, the œtiological concept propounded by one of us.[3] More and similar cases could doubtless be found by taking closer histories.

Finally, it must be stressed that biopsies may need to be repeatedly taken, as was done in the first case, before a lesion can be finally passed as being non-neoplastic.

We had previously concluded[3] that it seems useless to tell a man of 70 who has spent most of his life in the open to spend the rest of his days in the shade and that even this would not save him from the development of actinic cancer because the initiatory and sensitizing processes had already occurred. It seems reasonable, now, in view of the apparent multi-stage nature of both basal and squamous carcinogenic processes to advise our susceptible patients to avoid shaving with ordinary razors and to use electric razors so that minor cuts should not occur and this advice we are now giving at the Clinic. In addition, patients are warned to report early to their doctors if any small area of trauma, however trivial, should fail to heal within 2-3 weeks. For the first time we can offer some positive practical suggestions to reduce the incidence of facial cancers—the commonest cancer of the White man in South Africa.

SUMMARY

1. Cases of different neoplasms developing after trauma on the face of men who had been exposed to sunlight for many years are reported.

2. In 2 cases a basal-cell carcinoma developed on the site of an injury. This response is very rare.

3. The occurence is explained on the basis of the theory of multi-stage carcinogenesis.

4. A positive simple suggestion is made which should reduce the incidence of facial cancer.

We wish to thank Dr. J. G. Burger, Medical Superintendent, Groote Schuur Hospital for permission to publish these cases, and Dr. J. M. Grieve and Prof. J. H. Louw, of the Departments of Radiotherapy and Surgery, respectively, for their interest. Dr. M. Sachs of the Department of Pathology is thanked for his interest and the reports.

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THE EFFECT OF AMINO-ACID SUPPLEMENTATION ON THE NUTRITIONAL VALUE OF MAIZE PROTEIN FOR HUMAN ADULTS*

A. S. TRUSWELL, M.B., CH.B. and J. F. BROCK, M.D., F.R.C.P.

C.S.I.R. Clinical Nutrition Research Unit, University of Cape Town and Groote Schuur Hospital

The nutritive value or quality of a protein must ultimately be defined by the results of biological testing in the species of animal concerned. The better the nutritive value the higher the proportion of a protein that can be used for tissue synthesis in a person to whom it is fed. Conversely, the poorer a protein, the more of it is not used and excreted mainly as urea. Tissue synthesis in adults usually consists of replacement of wear-and-tear break-down.

* Abstract of a paper read at a meeting of the Research Forum, University of Cape Town, held at Groote Schuur Hospital, Cape Town, on 10 December 1958.

Since the biological value of proteins ranges from 0 to 100%, it follows that protein quality is very important when the quantity eaten is limited or body losses are increased.

It is generally accepted that the nutritive value of a protein depends on its amino-acid composition (aminogram). Its utilization, when fed to man, can be predicted by comparing its aminogram with the minimum amino-acid requirements of human adults. These have been determined by Rose[1] and others. Eight amino acids are essential and the proportional requirements range from 1 for tryptophan to 4½ for phenylalanine and leucine. For maximum utilization a dietary protein should supply amino-acids in the same proportions.

There are 4 reasons why protein quality estimated from the aminogram is not always borne out:

(1) The requirements were measured under artificial conditions. The basic pattern of amino acids was arbitrarily chosen. The calorie intake had to be raised to achieve nitrogen balance on pure amino-acid mixtures. Some of the amino acids were given in the DL- form.

(2) Natural proteins differ in digestibility and therefore availability of their constituent aminoacids.

(3) In animals adequately provided with all essential amino-acids, excess of one may impair the nutritive value of the dietary protein.[2]

(4) Published aminograms for any food vary quite widely. The reasons for this are partly technical but much depends on variety, climate, soil conditions and processing.

In this country we are interested in maize, which is the staple cereal of the Bantu. Where animal protein is scarce maize is likely to provide the bulk of the protein intake. For animals the biological value of whole maize appears to be somewhat lower than for wheat.[3] Very few tests have been made in man. The existing reports are of little value. They are either based on observations over a short period in a single subject or the maize used was highly extracted (e.g. corn flakes).

Aminograms of maize show deficiencies of tryptophan and lysine. The rather low methionine content is probably not critical; it is no lower than in casein which Cox *et al.*[4] could not improve for human adults by methionine supplementation. The leucine content of maize is high. Harper *et al.*[5] found that excess leucine impairs the growth of rats but not if extra isoleucine is given.

In the work reported here normal human male adults were studied in a metabolism ward. Their nitrogen balances were compared when they were fed on maize meal alone and on maize supplemented with amino acids and when half the maize protein was replaced by milk or casein. There were different basal conditions in the separate experiments, partly to see if any of these influenced sensitivity to differences of protein quality. At least 6 days were allowed for adjustment before comparisons were started. The protein and calorie intake and other conditions were kept constant in each experiment. Thus, in each test the subject was his own control. The diets were alternated every 9 days while the subject remained on continuous balance.

The maize used was a single batch of 'straight run' (97% extraction) South African white mealie meal. It was kept at 32-40°F until a week before it was eaten. The protein intake was usually about 1 g. per kg. per day. In most experiments maize provided over 90% of the protein and the rest was from pumpkin. The amino-acid supplements were tryptophan and lysine, with and without isoleucine. Together they replaced 5% of the nitrogen intake. Extra vitamins, iron and calcium were given.

Results

1. In 4 out of 5 subjects the nitrogen balance was negative on maize at an intake of 0·5 g. protein per kg. per day. On maize at 1 g. protein per kg. there was a small positive balance (less than +1 g. N per day) in 4 out of 6. When, at this intake, half the maize protein was replaced by milk or casein, nitrogen was retained by 4 out of 4 subjects.

The F.A.O. Committee on Protein Requirements[6] concluded that 0·35 g. per kg. of a good quality protein is sufficient for adult maintenance. Compared with this, maize is inferior.

2. Supplementation of maize with tryptophan, lysine and isoleucine produced a more positive nitrogen balance in 12 out of 12 experiments on 8 out of 8 subjects. This improvement is statistically significant and based on a total of 359 balance days. There was a tendency for a smaller faecal nitrogen on the amino acids. The difference was still significant when corrected for this and for the order (in the experiment) in which amino-acid supplementation was given.

3. In 4 out of 4 subjects maize supplemented with tryptophan and lysine (without isoleucine) has given a higher nitrogen balance than maize alone. The improvement with these 2 amino-acids was nearly as much as when isoleucine was also given.

The following incidental observations were made:

4. Four subjects were on 1 g. protein/kg. under static conditions. All had had negative nitrogen balances initially, indicating adjustment to a reduced intake. On maize the balance was positive in 3 but increased in all when amino acids were given. These and other data suggest that adults in nitrogen equilibrium have balances nearer +1 g. a day than 0. Other workers who have observed normal adults for a long time have found the same.[7] This phenomenon can be largely explained by unmeasured cutaneous nitrogen losses. Allowance must be made for it in estimating minimum protein requirements.

5. In 2 experiments addition of sugar to the diet produced a gain in weight and increased skinfold thickness. At the same time the nitrogen balance increased considerably. It returned to its former level when the extra sugar was withdrawn. The largest differences between the nitrogen balance on maize with and without amino acids were seen in experiments where the calorie intake appeared to be more than adequate.

6. In an attempt to enhance the difference between nitrogen balance on maize and maize with amino acids, 2 subjects were given testosterone derivatives. One received 25 mg. 'Durabolin' intramuscularly every 3rd day, the other 100 mg. norethandrolone ('Nilevar') orally a day. The nitrogen retentions were unimpressive. It is suggested that this happened because dietary protein was restricted in quantity and quality.

The results with amino-acid supplementation agree very well with provisional reports by Scrimshaw *et al.*[8,9] These workers found that children with kwashiorkor fed on corn masa had improved nitrogen retention when lysine and tryptophan were added. Isoleucine produced further improvement but methionine did not.

The early experiments were initiated by Dr. E. B. Dowdle. This work was supported by a grant from Messrs. E. I. du Pont de Nemours.

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SOUTH AFRICAN MEDICAL AND DENTAL COUNCIL: ADDRESS BY PRESIDENT
SUID-AFRIKAANSE MEDIESE EN TANDHEELKUNDIGE RAAD: REDE DEUR DIE PRESIDENT*

PROF. S. F. OOSTHUIZEN, M.D. (CAPE TOWN), F.R.C.P. (EDIN.), D.M.R., F.F.R.

Dit is met 'n gevoel van innige waardering dat ek u hiermee wil bedank vir die groot eer my aangedoen deur my te herkies tot President. Ek is diep onder die indruk van die eer en sal na die beste van my vermoë poog om die vertroue wat u in my gestel het, te handhaaf. Dr. Karl Bremer het by sy verkiesing as President van die Raad gesê dat dit die grootste eer was wat hom

* Address delivered at the 68th meeting of the South African Medical and Dental Council, Pretoria, January 1959.

ooit te beurt geval het. Dr. Radford het dikwels gesê dat lidmaatskap van die Raad by hom net so swaar weeg as lidmaatskap van die Volksraad.

Dis vandag 'n geleentheid waar ons nuwe lede verwelkom, maar ou lede nie vergeet vir hulle bydrae nie. Terselfdertyd, by die verwelkoming van die nuwe lede, wil ek graag melding maak van die uitstaande dienste wat gedurende hulle ampstermyn deur lede wat nie vandag by ons is nie, gelewer is, te wete drs. Bloom, Braun, Breyer, Graham, Loubser, C. Shapiro, M. Shapiro, van Schalkwyk en Vermooten. Hulle was sonder uitsondering waardevolle lede van die Raad en elk het op sy gebied 'n bydrae gemaak tot die werksaamhede van die Raad. Ons dink aan hulle vandag en sal hulle ook nie in die toekoms vergeet nie. Sonder uitsondering het hulle die Raad en sy werk leer liefkry en waardeer soos ek oortuig is dat dit ook met al die nuwe lede sal gebeur. Ek wil ook graag melding maak van mnr. William Impey wat nou afgetree het as Registrateur. Hy het die Raad baie jare lank met ywer en toegewydheid gedien en het diep spore hier nagelaat.†

Membership of this Council is a great honour, but it places great responsibilities on the shoulders of each and every member. The Council as a statutory body and charged with wide powers has a great duty to discharge in the interests of the public and the professions. This is not the occasion to go into detail about the numerous duties and responsibilities of the Council but it is perhaps opportune to refer briefly to some of the more urgent ones:

1. *The Medical and Dental Curricula*

The appropriate committee of the Council is at present dealing with this matter and a report will be made to the Council in due course. It is not only necessary to lay down reasonable minimum standards, but also to deal with the immense problem of overcrowding of the curriculum with the masses of detail which result from the ever-forward marching of medical and dental progress. In addition there have been introduced all the implications of the atomic age where startling developments take place with an ever-increasing tempo. As a result of the deliberations taking place at present, important amendments to regulations for medical and dental students may become necessary.

2. *Complaints against Registered Persons*

The usual procedure is that these complaints are carefully considered by the Executive Committee and then reported to the Council. The end result is usually either no action or an enquiry. In my opinion the Council will have to give serious consideration to finding some formula to deal with cases between these extremes.

3. *Amendments of Act and the Regulations*

(i) At the next meeting of the Council consideration will have to be given to the question of amendment of the relevant section of the Act and the regulations framed thereunder, to make it possible for interns and postgraduates from other countries to work at our institutions for specified periods for the mutual benefit of all concerned, and in some small way to repay the hospitality which our graduates have received through the years at overseas institutions.

(ii) *Section* 34 *of the Act*. Amendment of this section, dealing with persons not registered by the Council as medical practitioners practising for gain, is overdue. As the section now stands it is very difficult for the ordinary courts of the land to return a finding of guilt. The section is in fact full of loopholes and provides a veritable paradise for quacks to exploit the credulity of the public, often to the great detriment of the public.

(iii) *Rules for Specialists*. The Council has already decided that the Specialists Register should be maintained with certain restrictions. In drafting the rules and embodying these restrictions a number of difficulties and snags have been encountered, which have made it impossible to submit these rules for promulgation. The *ad hoc* Committee to be appointed will be faced with these difficulties and it is hoped that the entire matter will be settled early during the life of this Council.

The Specialist Register has undoubtedly done a good deal to raise the general standard of specialist practice, but the serious danger of over-regulation and interference with the legitimate practice of medicine has loomed on the horizon.

(iv) *Rule* 19. Proposals for the amendment of rule 19 and 19 (*bis*) have been considered by the Council and in fact it has been decided that the amended rules 19 and 19 (*bis*) should be referred to the Minister for approval and promulgation. Additional sub-rules 19 (*quat*) and 19 (*ter*) have been evolved and have not yet been finalized. Rule 19 (*ter*) dealing with practitioners in the employ of corporate bodies, and where an attempt has been made to obtain some measure of indirect jurisdiction over such corporate bodies through the medical or dental men in their employ, appears to be *ultra vires* according to legal opinion just received.

(v) *Supplementary Health Services Bill*. The policy of the Council at present is still compulsory registration, and successive Councils have endeavoured to influence the Government of the day to enact legislation to achieve that. Thus far it has not been found advisable to do so, because of the contentious nature of such legislation. The Council has now received the considered views of the Medical Association of South Africa and this Council will shortly have to deal with this matter once more.

4. *National and International Cooperation*

It is hoped that the new Council will pursue the policy of the previous Council of close national and international cooperation. In the national field it cooperates actively with the Medical Association of South Africa, the Dental Association of South Africa, the Department of Health, the Provincial Administrations and other statutory bodies like the South African Nursing Council, etc. In the international field the Council is concerned with other Medical and Dental Councils, International Medical Associations and our Liaison Officers in Great Britain and the United States of America.

In this rapidly shrinking world, owing to the impact of new methods of travel and communication, the time of educational and scientific isolation is past. This Council can play an increasingly important role in promoting improved national and international relations, in rendering a service to mankind in diverse ways, including education, and ensuring a high ethical code.

This Council has to deal with many vital facets of the national life of the nation and therefore has to face innumerable problems. I have sufficient confidence in the Council to feel sure that the contribution will be worth while and that the Council will face up to these problems.

Medicine, notwithstanding all these new developments, is a warm personal science (dentistry being specifically identified with it). In this era of technology we must not be swamped or overrun, but remain loyal to those traditions of honour and good professional behaviour of which we are the custodians and in which the history of medicine is so deeply steeped.

† The President then proceeded to introduce the new members of the Council and he also welcomed Mr. W. H. Barnard, who assumed duty as Registrar of the Council on 1 January 1959, and Mr. J. H. de Jager, who assumed duty as Assistant Registrar on the same date.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES REMOVED FROM THE LIST

The name of the following medical aid society has been removed from the list of approved medical aid societies as from 1 January 1959:

Stewarts and Lloyds' Medical Aid Society, P.O. Box 74, Vereeniging.

MEDIESE HULPVERENIGINGS VAN DIE LYS GESKRAP

Met aanvang 1 Januarie 1959 is die naam van die volgende mediese hulpvereniging van die lys van goedgekeurde verenigings geskrap:

Stewarts and Lloyds' Medical Aid Society, Posbus 74, Vereeniging.

The society has written requesting medical practitioners to submit all accounts for services rendered to members of the Society up to 31 December 1958 not later than 31 March 1959.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
21 January 1959

Die hulpvereniging het geskrywe om te vra dat geneeshere alle rekenings vir dienste aan lede van die hulpvereniging tot op 31 Desember 1958 gelewer, vóór 31 Maart 1959 moet indien.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
21 Januarie 1959

IRON-DEFICIENCY ANAEMIA IN CAPE COLOURED AND AFRICAN CHILDREN IN CAPE TOWN

CORRECTIONS

In this article by Drs. Philip Lanzkowsky, M.B., Ch.B. (Cape Town) and David McKenzie, M.B., Ch.B., M.Med. (Path.) (Cape Town), which appeared on page 21 of the issue of the *Journal* of 10 January 1959 (**33,** 21), the following corrections are to be made on page 23

1. *Fig.* 5. The indication of the type of treatment given should read as follows (in this order):
On 4 ml. Parrish's food per day ————
After 16 ml. Parrish's food per day .....
After 1·35 g. Ferr. et ammon cit. ·—· ·—· ·—· ·—·
Wintrobe's Standard —— —— —— —— —— ——

2. *Table VII.* The first part of the last line should read as follows:
All cases combined: $p > 0{\cdot}05$ i.e. not significant.

3. *Table IX.* The 3rd line should read '% Anaemic' not 'Anaemic'.

4. *Page* 23, 2nd column, lines 2 and 3 should read as follows:
'. . . in 132 Bantu school children on the Rand was 97·48% and concluded that the average Bantu school child did not suffer from anaemia'.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. C. Frank, after practising for nearly 20 years at Porterville, Cape, has now been appointed Senior Medical Officer at the Boksburg-Benoni Hospital. Dr. Frank took up his new appointment on 2 January 1959.

* * *

South African Paediatric Association. A meeting of the Cape Town Sub-group of this Association will be held on Tuesday 3 February in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape, at 8.15 p.m. Dr. Harold Cooper will speak on 'Psychological aspects of congenital heart disease'.

* * *

Research Forum, University of Cape Town. A meeting of Research Forum will be held on Tuesday 10 February at 12 noon in the large A-floor lecture theatre, Groote Schuur Hospital, Cape Town. Dr. C. S. Jones will speak on 'Clinical trials of a new barbiturate' and Dr. A. B. Bull will speak on 'Blood loss in infants and small children during surgery'. All interested are invited to attend.

* * *

Red Cross War Memorial Children's Hospital, Rondebosch, Cape. The next meeting in the Postgraduate Seminar Series of lectures held under the auspices of the University of Cape Town, Department of Child Health, will be held in the lecture theatre of this hospital on Wednesday 4 February at 5 p.m. Prof. F. J. Ford will speak on 'Resuscitation of the newborn infant'. All practitioners are welcome.

* * *

Mr. B. W. Franklin Bishop, F.R.C.S. (Edin.) of Durban has been elected President and Secretary of the Association of Plastic Surgeons of Southern Africa for the ensuing year. The present full members of the Association are: Mr. J. B. Cuthbert, F.R.C.S. (Edin.) of Johannesburg; Mr. D. S. Davies, F.R.C.S. (Edin.) of Cape Town; Mr. D. Davies, F.R.C.S. (Eng.) of Cape Town; Mr. J. Penn, F.R.C.S. (Edin.) of Johannesburg; and Mr. D. H. Walker, F.R.C.S. (Edin.) of Johannesburg.

* * *

Rehabilitation Association for Injured Workmen. A symposium on 'Surgery in abdominal trauma' will be held on Wednesday 11 February at 8 p.m. in the Gymnasium, 3rd floor, Brenthurst Clinic, Parktown, Johannesburg. The speakers will be: Prof. D. J. du Plessis on 'Trauma of the abdominal wall', Mr. W. Kark on 'Injuries to the abdominal viscera' and Mr. C. D. Kisner on 'Genito-urinary injuries'. All doctors are cordially invited to attend and join in the ensuing discussions.

* * *

Cape Midlands Branch (M.A.S.A.). The annual dinner of this Branch was held recently and was attended by their Worships the Mayors of Port Elizabeth and Walmer, the System Manager of the South African Railways and the Chairman of the Hospital Board. During the evening the Bronze Medal of the Medical Association of South Africa was presented to the Hon. Secretary of the Branch, Dr. P. Jabkovitz, by the President of the Association, Dr. R. Schaffer, who made a special visit to Port Elizabeth for this occasion. The floating trophy of the annual Medical and Dental Golf Competition was also presented to Mr. R. C. B. Macrae during the dinner. Mr. Macrae has won this trophy on 3 previous occasions.

* * *

Dr. J. J. van der Wat, M.B., B.Ch., Dip. O. & G. (Rand), voorheen van die Algemene Hospitaal, Johannesburg, praktiseer nou as spesialis in Verloskunde en Vrouesiektes te Listergebou 602, Jeppestraat, Johannesburg. Telefone: Spreekkamer 22-9919, woning 42-8376, noodoproepe 22-4191.

Dr. J. J. van der Wat, M.B., B.Ch., Dip. O. & G. (Rand), formerly of the General Hospital, Johannesburg, is now practising as a specialist Obstetrician and Gynaecologist at 602 Lister Building, Jeppe Street, Johannesburg. Telephones: Rooms 22-9919, residence 42-8376, emergency 22-4191.

IN MEMORIAM

LOUIS NAHUM KAPLAN, M.R.C.S., L.R.C.P.

Dr. R. Rabinowitz of Pretoria, writes: Dr. Louis Nahum Kaplan passed away on 23 December 1958 at the age of 45 years.

He was born in Stellenbosch and after matriculating in that town, went overseas to study medicine. He received his M.R.C.S., L.R.C.P. degrees at Guy's Hospital, London in 1936.

Dr. Kaplan

On his return to South Africa he first practised at Richmond, Cape, and during this period he married Miss Sylvia Goldstein of Pretoria. He moved to Pretoria in November 1940 and remained there in general practice.

He was an ideal example of the family doctor and if he attended to members of one family was quickly 'adopted' by other branches of the family.

Dr. Kaplan was a skilled clinician and a wise counsellor to his patients, but what endeared him to those who knew him was his excellent sense of humour and winning personality. He was loved by all his patients, whether rich or poor, and of all creeds and races.

He had the misfortune to suffer from a progressive and unrelenting illness. In the last few years his health was failing and he faced his sufferings and sorrows with a fortitude which amazed all those who knew him.

It can truly be said that the medical discoveries of the last decade, ranging from antibiotics and cortisone to new urological techniques and the artificial kidney, helped to prolong his life. He was forced to retire from general practice in October 1958 and he hoped to pass the days doing things which a busy general practice had previously denied him. Little did he realize how short was to be the span of his days.

To his wife, son and daughter go our heartfelt sympathies.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

TITANIUM IN SURGERY

Titanium is a relatively new metal. For some time stainless steel and cobalt-chromium alloy have been used extensively in surgery for the internal fixing of fractures and the replacement of diseased sections of bone, but the light-weight and high-corrosion resistance of titanium, coupled with its considerable strength, has led to experiments being carried out in 'surgical repairs' with excellent results. The main advantages of titanium over other metals and alloy systems in the field of surgery are:

1. It has a high tensile strength and low weight.
2. Ductility and impact values exceed the safety limits.
3. It is not easily attacked by corrosive media and is unaffected by tissue fluids.
4. It becomes adherent to the bone in which it has been fixed and no reaction has been detected.
5. The metal is translucent to X-rays.
6. No solution of the titanium occurs.

Manufacturers: Down Bros. and Mayer and Phelps Limited.

Full particulars and instruments all obtainable from: Westdene Products (Pty.) Ltd., at Johannesburg (23 Essanby House, 175 Jeppe Street), Cape Town (408 Grand Parade Centre, Castle Street), Durban (66 67 National Mutual Building, Smith Street), and Pretoria (210 Medical Centre, Pretorius Street).

PHARMACEUTICAL NEWS : FARMASEUTIESE NUUS

SKF LABORATORIES (PTY.) LTD.: GRANT FOR PLANT RESEARCH

The council for Scientific and Industrial Research have received a grant of £3,500 for a study of South African plants to determine whether they contain chemical agents which might be valuable as new drugs. The grant was made by Smith Kline and French Laboratories, Philadelphia, USA, parent firm of SKF Laboratories (Pty.) Ltd., Port Elizabeth.

Although several studies have been made of the rich South African flora, these have largely concerned the toxic effects of native plants on animals. The C.S.I.R. study, however, will be orientated towards identification of chemical substances which might have useful therapeutic properties in humans. Special attention will be directed to substances traditionally used in local folk medicine.

In research of this sort, plants are first identified botanically. Then chemists isolate the plant constituents, which in turn are tested by the pharmacologist for medicinal properties. Promising drugs are tried in the clinic.

Although more and more pharmaceutical agents are made synthetically, scientists are again turning their attention towards plants as possible sources of new drugs. For example, one class of tranquilizing agent was developed from studies of a snake-like root which has been used in India for centuries.

Another factor in this search is that once chemists have extracted and identified medically active substances, it is frequently possible, in the process of synthesis, to reproduce the molecule of the chemical and to alter the molecular structure so that undesirable side-effects are eliminated in whole or in part.

The parent firm of SKF Laboratories (Pty.) Limited is engaging in similar studies of plant life in other regions of the world.

MEDICAL FILM PROGRAMME

A 30-minute motion picture dealing with the world-wide problem of cross infections in hospitals will be produced jointly by the American Medical Association, American College of Surgeons, and the American Hospital Association. This timely and basically important film has been made possible by the cooperation and support of Johnson and Johnson of New Brunswick, New Jersey.

The film, which will be in sound and colour, is designed to educate all levels of hospital personnel concerning the many avenues by which infection is spread throughout a hospital and it will utilize the staphylococcus by way of illustration and as an example of one of the most important phases of the problem.

Produced under the supervision of Dr. Carl Walter of Boston, Associate Clinical Professor of Surgery, Harvard Medical School, one of the recognized pioneer investigators in this field, and a committee representing the AMA, ACS and AHA, it will deal with the broad fundamentals of phases of the problem and lay the groundwork for the delineation of the problems relating to specific fields in a series of shorter films to follow.

The presentation will have its première showing at the annual meeting of the American Medical Association in Atlantic City next June, again at the conclave of the ACS in October and subsequently at professional gatherings throughout the world.

The production of this film will be coordinated by Ralph P. Creer, Chicago, Director of Motion Pictures and Medical Television of the American Medical Association.

NEW AGENCY FOR PETERSEN LIMITED

Petersen Limited has been appointed sole distributor in the Union of South Africa for the pharmaceutical products of Ed. Geistlich Sons Ltd., Wolhusen, Switzerland. The pharmaceutical division is a production branch of the firm of Ed. Geistlich Sons Ltd., founded in 1850 for the manufacture of chemicals, with works at Schlieren and Wolhusen.

Present-day production of the 'Gewo' medical specialities is based on the work of their own research scientists whose task and aim is to place in the hands of the physician, medicaments which are free from every imperfection. Thanks to its enlightened attitude towards many of the problems which face us today, the firm of Geistlich occupies its assured place among the chemical and pharmaceutical undertakings in Switzerland and abroad.

During the initial introductory period Petersen Limited will concentrate on 2 important products. In introducing these products Petersen Limited supply the following information:

1. **Dipasic** (Isonicotinic—acid—hydrazide—p-aminosalicylate).

This product functions in a manner similar to that of the former INH/PAS swing therapy and possesses in addition various advantages in tolerability and ease of administration. *In vitro* tests show that Dipasic is fully effective not only against sensitive strains but also against strains resistant to PAS/INH and streptomycin *in vitro*. Clinical tests have confirmed these bacteriological results.

Indications. Dipasic can be used for all forms of tuberculosis. Satisfactory results have been obtained in pulmonary and extrapulmonary tuberculosis. Children, in particular, respond to Dipasic therapy and the need for streptomycin injections is eliminated.

Dosage. The dose level is dependent on the clinical condition, but in general, the following dose levels are recommended:

Adults: 1-2 tablets (i.e. 100-200 mg.) per 10 kg. body weight daily.

Children: 2-3 tablets (i.e. 200-300 mg.) per 10 kg. body weight daily.

These dose levels are spread over the day in 3-4 doses. It is, of course, not possible to reduce the duration of treatment to a scheme as each patient must be treated according to individual requirements.

Presentation. Dipasic is available in containers of 100, 500, 1,000 and 5,000 tablets each containing 100 mg.

2. **Gewodin.** This is a completely new formula of an analgesic/antipyretic preparation which is an innovation in the field of such remedies. The formula for Gewodin is as follows:

3-(N-methyl-(a-methyl-B-phenil)-ethyl-amino-methyl)-4-isopropylnorantipyrin	0·025 g.
Dimethylaminoantipyrin	0·100 g.
p-Acetophenetidin	0·200 g.
Caffeine	0·030 g.

Indications. Gewodin is indicated for the treatment of symptoms due to weather changes, headaches—migraine, rheumatic pains—neuralgia, dysmenorrhoea, toothache; colds and chills—influenza.

Tolerance. There are no side-effects referred to the gastro-intestinal tract or to the circulatory system, nor are there any allergic manifestations even in patients who have taken several tablets a day (3-5) for several weeks.

Dosage. Adults: 1-2 tablets up to 3 times daily. Children: ½-1 tablet daily.

Presentation. Gewodin is available in boxes of 10 and 100 tablets and in hospital packings.

Further details concerning these products may be obtained from the distributors or from any wholesale supplier.

PAM FOR INSECTICIDE POISONING

Some misunderstanding arose as a result of the last sentence of our Editorial[1] on 'Poisoning with pesticides', published on 13 December 1958, which reads as follows: 'Professor Steyn urged that it was important to make PAM available in South Africa without delay'. M.L. Laboratories (Pty.) Ltd. have imported PAM and made it available to Prof. Douw G. Steyn and all the hospitals in the Transvaal free of charge.

1. Editorial (1958): S. Afr. Med. J., 32, 1189.

BOOK REVIEWS : BOEKRESENSIES

YEAR BOOK OF RADIOLOGY

The Year Book of Radiology 1957-1958. Radiologic Diagnosis. Edited by John Floyd Holt, M.D. and Fred Jenner Hodges, M.D. *Radiation Therapy*. Edited by Harold W. Jacox, M.D. and Morton M. Kligerman, M.D. Pp. 445. Figures 335. $10.00. Chicago: Year Book Publishers, Inc. 1957.

The diagnostic section, as usual, provides a wide review of the year's papers from journals received between June 1956 and June 1957. Radiation hazards, being the most discussed subject in this field today, earns a sober 4-page critical review by the editors and can be regarded as authoritative.

Under 'Technical Developments' are summarized useful papers on 'A Double Mirror for Stereoscopy', 'Log Etronography' (for re-conditioning over-exposed films) and a new technique for reproduction by solarization using out-dated film.

On the clinical side it would be invidious to single out interesting sections, still less individual papers. What must impress any reader is the extensive use to which isotopes are put in diagnostic radiography overseas, and how meagre, if not altogether absent, the facilities in the Union are. Examples are the localization of renal calculi during surgery by portable radiography, using Thulium-170, and demonstration of the shape and structure of the liver, using radioactive tetraiodophenolphthalein or colloidal gold.

Since the price of this book was 37s. in 1947, 64s. 9d. in 1951 and the price at present is 92s. 6d., the book may soon be beyond our reach.

Abstracts from most of the important articles in the field of radiotherapy from June 1956 to June 1957 are contained in this volume, ably edited by Jacox and Kligerman.

In a pertinent introduction to the therapeutic section, emphasis is laid on the need to keep a proper balance between beneficial and harmful effects of irradiation, and as the editors say 'the confusion between the exposure to fall-out radiation of large population groups and the ill patient who must have diagnostic or therapeutic radiation is quite unfortunate'.

Every aspect of the subject is covered and a number of interesting articles are included giving an honest evaluation of various methods of treatment. There is also much of interest and real value in the radioisotope radiobiological and dosimetry sections.

Steady advances are being made in the application of very high voltage X-irradiation and cobalt teletherapy, and techniques are being perfected. Although the advent of these high energy radiations has not markedly improved the cure rates, yet very encouraging results are being obtained in certain lesions.

It is sad to reflect that we have still not availed ourselves in this country of these very real advantages that every radiotherapist knows.

I.M.S. and L.W.

ORTHOPAEDICS FOR THE GENERAL PRACTITIONER

Orthopedics for the General Practitioner. By William E. Kenney, M.D. and Carroll B. Larson, M.D., F.A.C.S. Pp. 413. 180 Illustrations. South African Price: £5 2s. 0d. St. Louis: The C. V. Mosby Company. 1957.

The question of whether the approach of a general practitioner to a medical problem should be any different basically from that of the specialist is one which often occupies the minds of medical teachers today. Is there, in fact, such a thing as medicine for the general practitioner, or is there only medicine?

It is generally assumed that an undergraduate training should fit a doctor for general practice and it therefore follows that undergraduate text-books should supply the needs of the general practitioner.

In *Orthopaedics for the General Practitioner*, however, the authors have produced a book which is neither an undergraduate text-book nor a book for the specialist, but is truly designed with the prime object of aiding the family doctor in the diagnosis and management of orthopaedic clinical problems. This does not mean, however, that the book is not a complete, well-written and up-to-date account of the subject. It is in its approach rather than in the subject matter that it is different.

The authors have listed, in age groups, all the orthopaedic conditions which affect each anatomical area of the body so that the general practitioner, presumably knowing the patient's age and the site of his complaint, simply turns to the correct page to find the diagnosis and treatment.

The result of this arrangement is that although the book is of value as a reference book, it lacks continuity and fails to convey the principles of the subject and, therefore, any overall picture of orthopaedics to the reader.

The book is well produced and illustrated with photographs and diagrams and it includes such items of value as advice on filling out insurance claims, assessing and estimating degrees of disability and permanent disablement, and even gives advice on one's approach when discussing such orthopaedic problems as cerebral palsy and poliomyelitis with a child's parents.

This book will be of value to many doctors in general practice.

T.L.S.

TUMOURS OF THE SKIN

Tumors of the Skin. By Herbert Z. Lund, M.D. Pp. 330. 265 Figures, 1 Color Plate. $3.00. Washington: Armed Forces Institute of Pathology. 1957.

This book deals with the histology of tumours of the skin, excluding melanomata, but including tumour-like hyperplasias and malformations, and it is highly recommended for all who have to deal with these conditions.

There is an interesting discussion on the classification of cutaneous tumours, followed by descriptions of the individual lesions. A very large proportion of the available space has been devoted to black and white microphotographs. These are excellent, but the one colour plate whets one's appetite for more. It is to be hoped that the printing industry will soon make a big contribution to modern medicine by reproducing colour photographs cheaply.

Binding costs have been eliminated by stapling the pages together. At the same time provision has been made for the use of a loose leaf binder.

S.S.

PRACTICAL ALLERGY

Practical Allergy. By M. Coleman Harris, M.D., F.A.C.P. and Norman Shure, M.D., M.S., F.A.C.P. Pp. xiii+471. 22 Figures. 60s. 9d.+1s. 9d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

This work represents a summary of ideas and practice in the diagnosis and control of allergic diseases. Controversial aspects of this complex subject are avoided and the authors have designed a 'simple text-book which the doctor can use as a "vade-mecum".'

The information in the book is elementary knowledge to the practising allergist and is available to him in much greater detail in any of the standard text-books on allergy which would similarly be used by a senior medical student seriously interested in learning about the subject.

The general practitioner, however, may find the reading of this book useful to obtain in compact form a general idea of the problems, methods and tools in allergy problems and perhaps also to find some guidance in the handling of his allergic patients.

D.O.

SUDAN MEDICAL SERVICE

The Sudan Medical Service. An Experiment in Social Medicine. By H. C. Squires, C.M.G., D.M., F.R.C.P., D.P.H. Pp. xii+138. Illustrations. 15s. net. London: William Heinemann—Medical Books—Ltd. 1958.

This is a factual history of the Sudan Medical Service—its establishment and growth. As writer, Dr. Squires is specially qualified by virtue of his 43 years service almost from its inception.

After the final battle at Omdurman in 1899, essential medical services were the duty of the army of occupation. The Sudan Medical Service became autonomous in 1904, but did not include the southern part until after World War I. This Service is traced from its earliest, personal pioneering days, through periods of rapid expansion, to its present comprehensive extent requiring almost £E3,000,000 annually to run. Interesting sections describe the epidemic and endemic diseases experienced.

The Service reached maturity during World War II when called on to play an important role in the Abyssinian campaign. Expansion thereafter was particularly rapid with its characteristic increasing medical specialization, and 'Sudanization'. The book is particularly important to the Sudan, but makes intructive and interesting reading for everyone.

H.R.A.

CORRESPONDENCE : BRIEWERUBRIEK

MEDICAL COUNCIL ELECTION

To the Editor: I shall be most grateful if you will allow me to express, through the medium of the *Journal*, my sincere thanks to the practising profession for the support given me in the recent election of the South African Medical and Dental Council. I am deeply sensitive of the high honour which has been bestowed upon me and am fully cognisant of my responsibilities as a member of the Council.

H. Grant-Whyte

232 King George V Avenue
Durban
16 January 1959

NEEDS FOR MEDICAL CARE

To the Editor: I welcome Dr. Susser's comments[1] on the article on clinical planning by Mr. Zwart.[2] His letter focuses attention on the importance of careful assessment of the needs of a population in regard to health services.

In the past there has been little planning of clinics and apart from the work of Professor Kark and the pioneer efforts of the Alexandra Clinic with which Dr. Susser was connected, this basic and important branch of medical science has not kept pace with similar advances in other medical fields.

Mr. Zwart is an architect and is concerned more with the design of clinics in relation to the population to be served than with the socio-economic factors affecting clinic attendances. The statistics presented are not fixed entities but are being continually revised. They have proved beyond doubt that a clinic conducted on the lines of Meadowlands can effect a considerable saving in hospital beds and in out-patient attendances. Satisfactory planning of the buildings has prevented congestion of patients with the premises—a serious problem in most clinics.

A new approach is being evolved with the integration of the various services dealing with health and disease. The shift of emphasis from the patient in the 'hospital bed' to the 'patient in his home' and in his own environment is a great step forward in helping to solve the complex problems of Bantu ill-health.

S. B. Sachs,
Johannesburg *Senior Medical Officer, Meadowlands Clinic*
9 January 1959 *Baragwanath Hospital*

1. Susser, M. (1958): S. Afr. Med. J., 32, 1240.
2. Zwart, W. (1958): *Ibid.*, 32, 1037.

THE ASSOCIATION'S FINANCES

To the Editor: It is with no small degree of amusement that I read your article 'The Association's Finances' in the *Journal* of 10 January 1959[1] in which a plea is made for financial aid to a struggling Medical Association.

It is correctly pointed out that costs have increased and, more important, that the pound is now worth only one-third of its value of 15 years ago. Therefore you justify a deficit and regard an increase in subscription from 2 to 6 guineas as very fair.

The same people (the administrators of the Medical Association) who put forward the above argument do not, however, deem it necessary for any rise in doctors' fees. Practically speaking there has been very little change in recent years.

May I suggest that if this is the logic of our present leaders they should be substituted by younger members in active practice who are in a better position to appreciate today's conditions and problems.

Wake up medicos and put your house in order.

V. Sorour

P.O. Box 642
Welkom
13 January 1959

1. The Associations' Finances (1959): S. Afr. Med. J., 33, 38.

[The fees paid by members of medical aid societies for consultations and visits by general practitioners have been nearly doubled since 1947 as the result of negotiations between the Association and the medical aid societies concerned. Private fees have been raised considerably by all the Branches.

Subscriptions for membership of the Association were increased by only £2 2s. 0d. a year.—*Editor.*]